DISEASES OF THE HEART.

DISEASES OF THE HEART;

THEIR

PATHOLOGY, DIAGNOSIS,

AND

TREATMENT.

BY

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PREFACE.

THE following pages have been in great part re-written, and the rest of the work has been carefully revised. This revision was required by the advances which have been made in our knowledge of the pathology and treatment of cardiac diseases since the first edition of this work was published. I have endeavoured, as in the former edition, to reduce into a practical form all that our researches have taught us concerning Diseases of the Heart.

I need scarcely say, that in the case of the treatment of acute cardiac diseases, as well as of all other acute diseases, a great change has been effected during the past few years. Modern diagnosis, pathology, and, above all, the telling truths of modern clinical medicine, have opened before us a new era in Therapeutics. Men still differ, it is true, concerning the reasons why this great change has been

effected in the practice of our art, but they are all tolerably well agreed in the conclusion: that the violent remedies applied by our forefathers in the cure of acute diseases are not the remedies which suit human nature in its present constitution.

Consequently, in the present edition, I have no longer had any difficulty in expressing freely my opinion as to the powerlessness of certain agencies, as, for instance, venesection and mercury in arresting inflammatory processes; nor in stating what I believe to be the true uses of those remedies in the treatment of diseases. A few years ago, it required a certain amount of moral courage to question the efficacy of the orthodox list of antiphlogistic remedies; but now, under the shelter of the authority of the very *patres conscripti* of our profession, men may express a rational scepticism in the orthodoxy of traditional medicine, without suffering condemnation as irreclaimable heretics.

And surely there is nothing surprising in the change which has thus passed into the practice of Medicine. The whole series of facts upon which rational Medicine is founded, have, during the past few years, been absolutely revolutionised; and necessarily, therefore, and logically enough, medical practice has likewise undergone a thorough reforma-

tion. We need not search about elsewhere for any violent hypothesis, by which to explain away the wonder of this novel phasis of Medicine. Scientific medicine, to be worthy of the term, must, manifestly, be based on scientific facts; and we are all agreed, that pathology, physiology, and anatomy, diagnosis and clinical medicine, are studies indispensable to the physician. If Medicine rested not on the facts which they impart, the art would have no foundation whatever; it would be a mere crumbling edifice.

Now, when we compare the pathology of to-day with the pathology of the past; the diagnosis of to-day with that of the past; the clinical study of disease as taught to-day and as taught a generation ago—surely we need not feel any surprise, that innovations should have found their way into a practice, which has so little accord with the light of modern researches.

When grave authorities pronounced that disease was to be evacuated at the mouth of the vein, and that inflammations could be jugulated or strangled in their birth by venesection, naturally enough bleeding was the optimist remedy. The practice of the physician was in good accord with his pathology. He was taught that an inflammatory entity had got possession of the affected part, that the tissues where

it had fixed its seat were in a blaze, and that the blood was the fuel which fed the fire; and the plainest logic drove him to the conclusion, that the way to put out the fire was to cut off its fuel—to bleed.

The practice of to-day must be attributed to our more enlightened pathology; and I cannot see that there is any necessity for calling in such an intervening deity as the change-of-type theory of disease to help us to an explanation of the fact.

My own firm belief is, that Medicine now, for the first time in its history, is entering into the path by which alone it can pass to the position of a science —if it be destined ever to reach that height. The fermentation which it has been lately undergoing was the process required for its purification—for the removal of the clogs and burthens of errors and crudities which centuries have gathered around it. Whilst this purifying process has been going on, ardent labourers occupied in its reconstruction and its establishment on an infallible foundation, have been gathering materials for the construction of the new edifice.

That we have had to lower our pretensions as curers of disease is only too certain. We no longer pretend to control and destroy diseases with the

master power which our forefathers imagined they possessed, nor do we now arrogate to ourselves those kind offices which nature, not our art, performs. We are at last arriving at a knowledge of the real and actual power which our remedies possess over the progress of diseases.

That Medicine has yet made but few advances towards the position of a science is lamentably true. The work to be done is all before us. Now, however, we are freed from the ignorance and prejudices which have for so many ages sustained the errors of Medicine, and overshadowed the practice of our art. Our knowledge, though very limited, is still, as far as it goes, *sure*. The line between that which is hypothetical and that which is demonstrable is clearly and well defined. We can mark where our positive knowledge ends, and estimate at their proper value the theories and the practices which we resort to and follow out in the cure of diseases. We have, in fact, what our forefathers had not, the knowledge of our fallibility and our limited powers; and it is just this, —the philosophic estimate of his actual powers over disease, — which distinguishes the rational and scientific physician of the present day from the vulgar empiric.

I have thought it right to make these few prefa-

tory remarks in order to anticipate the objection of any reader who might think that I had, in some instances, passed too lightly over that part of the subject which relates to the treatment of heart diseases.

W. O. MARKHAM.

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CHAPTER I.

PATHOLOGY OF PERICARDITIS.

ANATOMICAL DESCRIPTION.—PECULIARITIES OF THE INFLAMMATION.—ITS SPECIAL CHARACTERS.—EFFECTS OF THE PERICARDITIS ON THE DIFFERENT ORGANS OF THE BODY.—EFFECTS ON THE HEART ITSELF.—PERICARDIAL ADHESIONS.—EFFECTS ON ORGANS AND PARTS AROUND AND EXTERNAL TO THE PERICARDIUM: ON THE PLEURÆ AND LUNGS.—RELATION OF PERICARDITIS TO ENDOCARDITIS.—WHITE PATCHES (SPOTS) ON THE HEART.

PERICARDITIS consists in an inflammation of the whole or of a part of the serous membrane which lines the pericardial sac. It is not an idiopathic inflammation.* Occasionally we meet with it as the result of traumatic injury. Sometimes it is excited by the spreading of contiguous disease to the pericardial membrane, by the growth upon it of cancer or of tubercle, and by the opening into the sac of abscesses, hydatid tumours, &c. But it is most especially known to the physician as a local inflammation, associated with

* Modern pathology shows that idiopathic, as applied to local inflammations, is a false term. The so-called idiopathic inflammations are in reality local manifestations of a general affection, whose real nature is more or less obscure. A correct understanding of the term is clearly of every importance in the treatment of disease.

some special disorder of the system—with rheumatic fever, for instance, or pyæmia; in fact, as one of the local manifestations of some morbific influence which is operating generally in the body.

In this inflammation, as in the inflammation of other serous membranes, we note stages of congestion, of exudation, of absorption, of adhesion, of new-growth, and of organization. The quality or character of the exudation varies much, according to the nature of the formative impulse—of the condition of the body, and the nature of the disease—which presides over its deposition.

In rheumatic pericarditis, the exudation is chiefly of the fibrinous and serous kinds, and varies much in quantity. The serous part is, at one time, very abundant; at another, it is scanty—sometimes a few ounces, and sometimes several pints, being effused into the sac. The fibrinous layers again may be smooth and thin, or thick, uneven, flocculent, villous, and honeycombed, giving to the heart an aspect which older authors described as *cor villosum, hirsutum, hispidum, tomentosum.* Both the fibrin and the serum are occasionally tinged with blood, effused through rupture of new-formed blood-vessels. These exudations have a natural course to run. The fibrinous part, exuded from and adherent to the inflamed serous surfaces, gradually becomes organized; the serous portion undergoes absorption, and at last permits the fibrinous layers—the so-called false membranes—on the peri-

cardium, to come into immediate contact. Under such conditions, the opposed fibrinous layers gradually coalesce; and by their union and by the more perfect development of the fibrin and the growth of blood-vessels in it, a distinctly organized and vascular connexion (of areolar tissue) is established between the surfaces of the pericardium.

Purulent exudations into the pericardium are usually met with during the progress of serious affections of an adynamic type, such as pyæmia, tuberculosis, small-pox, puerperal fever, scarlatina. They have, for the most part, a fatal issue. Pus is a product incapable of a higher organization; but it has a tendency to undergo certain changes,—partially of absorption, and partially of degeneration,—or, as it has been barbarously termed, retrograde development. The fluid parts of it are absorbed, and the more solid parts of it gradually converted into cheesy, fatty, and calcareous, &c., matters. In this way are doubtless formed the "tuberculous," pultaceous masses, and the calcareous plates occasionally met with on the pericardium. The calcareous plates are sometimes so disposed as to convert the whole parietal layer of the pericardium into a hardened sac, forming thus the so-called ossified pericardium.

The pericardial inflammation affects, locally, both the heart itself and the parts—pleura and lungs—immediately surrounding the pericardium. Its influence upon the heart is of a twofold kind; it injures.

the organ *vitally* and *mechanically*, and both during the progress of the inflammation, and when the inflammation has passed away. At the onset the inflammation acts as a local irritant, and inordinately stimulates the heart's muscular contractions; but after a time, as always happens in the case of a function inordinately exercised, the vital power of the overwrought muscles is weakened, and the heart, in consequence, so to say, partially paralysed — the same result this as is sometimes noticed in the case of pleurisy and peritonitis, through their action upon the neighbouring intercostal and intestinal muscles. The muscular tissue of the heart, moreover, is in some cases permanently injured, through the action of the inflammation; and hence it is, that fatty and other degenerations of the organ become occasional consequences of pericarditis.

The action of the heart is also *mechanically* impeded by the pressure of the exudation upon it and its great vessels and upon the lungs. When the pericardium is much distended, this pressure acts with especial force upon the auricles of the heart. Solid fibrinous masses, also, deposited around the heart, necessarily, seriously interfere with its muscular movements. In cases of this kind the heart sometimes looks as if it had been pressed backwards and flattened by the pressure of the exudation, the auricles being thickly coated with fibrin, and crumpled down, and hardly to be distinguished from the ventricles by any line

of demarcation. I have also seen the aorta indented at one point from a like cause.* In such cases, moreover, we may conjecture, that the thick and hardened masses of fibrin accumulated around the base of the heart, interfere with the proper nutrition of the heart itself, by pressure on the coronary arteries. I think I have distinctly seen these vessels thus compressed.

The *remote* consequences of the inflammation are those functional derangements, &c. of the heart which result from adhesions of the pericardial surfaces and from the structural changes above mentioned effected in the tissues of the heart, whereby their elasticity and contractility have been impaired.

Authors differ concerning the effects on the heart of adhesions of its pericardial surfaces. The adhesions are said to occasion hypertrophy, hypertrophy with dilatation, dilatation, and atrophy of the heart. There is no doubt that each of these abnormal conditions of the organ may exist in coincidence with such adhesions, and it is not difficult to understand how this happens. When the heart's muscular structure has been left unimpaired, hypertrophy will result; but when its nutrition (after the fashion referred to) has been seriously injured, then we must expect the occurrence of atrophy or degeneration of its structures. Then, again, with pericarditis is very

* Here, perhaps, we have one explanation of the temporary systolic aortic bruit sometimes heard in pericarditis.

frequently associated endocarditis, and from endocarditis result defective valves, and from defective valves hypertrophy with dilatation of the heart. In considering the influence of the adhesions upon the heart, it is also necessary to take into consideration both the age of the adhesions and their extent. Dr. T. K. Chambers shows, in his *Decennium Pathologicum*, that death occurred at a much earlier period in those cases in which the adhesions were universal than when they are only partial. I must add, that cases of completely adherent pericardium are occasionally met with after death in which the heart appears to be, otherwise, perfectly healthy, and has perfectly performed its functions during life. From these considerations we may conclude, that the condition of the heart usually found associated with pericardial adhesions, depends rather upon the absence or presence of structural changes of its valves and muscular tissue than upon the adhesions themselves.

When the pericardial effusion is considerable, it interferes directly with the respiration by compressing the lungs; the pressure is especially exerted on the left lung, which, under such circumstances, is sometimes forced upwards and backwards, so as to be rendered, in great part, useless—its lower lobe being, in fact, more or less solidified. This impediment to respiration necessarily acts, indirectly, as an additional cause of obstruction to the already impeded action of the heart.

Pericarditis, when severe, extends by contiguity to the parts external to and immediately around the pericardium, exciting inflammation of the neighbouring pleura, and the pulmonary tissue. Exo-pericardial adhesions are the result — adhesions between the outer surface of the pericardium, and the borders of the lungs, and the adjacent walls of the thorax. Through the medium of these new adhesions—pericardial and exo-pericardial—the heart may become directly attached to the thoracic walls.*

Pericardial exudations, then, interfere with the circulation (and necessarily also with the respiration) in two ways: 1st, by the pressure exerted by them on the heart and its vessels, and by the injurious influence which they exercise over the nutrition of the organ; and, 2ndly, by their pressure on the lungs, preventing the free expansion of these organs.

Relation of Pericarditis and Endocarditis.—Endocarditis very frequently arises during the progress of rheumatic pericarditis; sometimes it precedes the pericarditis, and occasionally signs of the existence of both the inflammations are observed simultaneously. It is only in the case of rheumatic pericarditis, however, that we notice this connexion. Endocarditis, so arising, is to be regarded as the product of the

* It is under such conditions, as stated by Skoda, that we observe *retraction* of the intercostal space, during the heart's systole, at the situation of its apex.

same exciting morbid element as that which occasions the pericarditis.

Pathological anatomy has, hitherto, failed to throw any positive light on the exact nature of the serious and peculiar *cerebral disturbances* (of a maniacal and convulsive character) which are occasionally manifested during the progress of pericarditis. There is reason, however, to believe that they result from altered conditions (both qualitative and quantitative) of the blood. The brain when examined after death, in such cases, presents no signs of inflammation, but rather those signs which may be regarded as indicative of exhaustion. It is worthy of note that these nervous disorders appear to be less frequently observed now, since exorbitant venæsections have gone out of fashion.

White patches, or spots on the heart, are of very common occurrence, but they are of little or no clinical interest. They are formed of fibrinous materials in a low stage of organization, and appear to be of two kinds. In one sort, the fibrin is exuded *into* the tissue of the pericardium; in the other, it is exuded *upon* its surface. The fibrin thus deposited, is probably only exceptionally the product of inflammation; being in the majority of cases an exudation of unhealthy plasma, thrown out just as we find it in the abnormal white patches met with on the spleen, liver, and elsewhere. The fact of these white patches being frequently situated on the anterior part of the right ventricle, on the right auricle,

and sometimes on the posterior part of the heart, about the base of it, and over the coronary vessels, has not unreasonably induced writers to attribute them to the effects of friction, produced by the heart's movements. This view is corroborated by Dr. Jenner, who has pointed out the fact, that in rickety children, with deformed chests, "the chosen spot of the white patch is on the *left* ventricle, a little above its apex, at the spot which impinges against the fifth rib, where it projects inwards. Here it is evidently produced by attrition."

CHAPTER II.

CAUSES OF PERICARDITIS.

LOCAL CAUSES.—GENERAL CAUSES.—RHEUMATIC PERICARDITIS.—NON-RHEUMATIC PERICARDITIS.—RELATION OF PERICARDITIS TO RHEUMATIC FEVER.

In considering the causes of this inflammation, it is important that we should keep in view the fact already referred to, viz.: That pericarditis, excepting in those rare cases in which it is the result of traumatic injury, or is excited by contiguous disease, is not a mere local inflammation, but is the consequence of some exciting cause operating generally in the body.

The anatomical changes which represent the disease to us, therefore, are not, essentially, the disease. The changes are but the consequences of the unseen, but pre-existing agencies which are working in the system. And these agencies again, though pathology cannot clearly define their nature, are various; and they attack the vigorous, and they attack the weak; and certain of them operate in particular at one period of life, and some at another; and they fall upon other organs and parts of the body besides the pericardium. The same cause, for instance, which produces the rheumatic pericarditis is that which produces the rheumatic joints, and which produces

the endocarditis, and, it may be, the pneumonia and the pleurisy, which so often accompany pericarditis. And so again, the cause which, in disease of the kidney, excites inflammation of the pericardium, is the same which gives occasion to inflammation of other serous membranes and parts and organs of the body. Hence, then, the pericarditis is not the disease itself, but only one of its local accidents. It stands in the same relation to rheumatic fever as the local accidents of typhoid fever—viz., ulceration of Peyer's glands, pneumonia, or cerebritis—do to the essential element of the fever itself. What are usually called the causes of pericarditis, therefore, are, in reality, only co-existing manifestations of the self-same disorder as that which provoked the pericarditis. Correct views of the pathology of this affection are essential to correct views of its treatment.

For the better comprehension of the causes of pericarditis, the inflammation has been classed under two heads: 1st, Rheumatic pericarditis; and 2ndly, Non-rheumatic pericarditis. This division is of practical utility; for a study of the clinical history of pericarditis, in general, teaches us that there are marked points of distinction in its character, such as enable us readily to class the disease under one or other of these heads.

The cause of *rheumatic pericarditis* is, as I have already said, the cause which excites the rheumatic fever. Statistics have not yet given us any sure

information with regard to the frequency of pericarditis in rheumatic fever. M. Bouillaud and Dr. Hope found pericarditis present in about one-half of their cases of acute rheumatism. Dr. Taylor makes the number about one in nine; Drs. Budd and Latham make it about one in eight.

Results, however, on this subject, derived from the observation of cases which have been taken indiscriminately, without regard to age or sex, are of very little value; for it is a fact, that at certain periods of life the *coincidence* of rheumatism and pericarditis is much more common than it is at other periods. Thus, adult persons attacked by acute rheumatism are infinitely less liable to cardiac complications than the young are. After the age of thirty, pericardial and endocardial inflammation are, comparatively, rarely met with as the associates of rheumatism; whilst, on the other hand, before the age of twenty, and in early life, they are more frequently present than absent, in connexion with that disease.

Observation, indeed, shows: that rheumatic pericarditis attacks those of weak rather than of strong constitutions; that it is more common in the delicate and young, than in vigorous persons at the prime or middle periods of life; and that the degree of inflammation—that is, the general febrile reaction and the local exudation—is also greater in them than in the strong; and that it is often especially severe in young

females. Climate, also, may have something to do with the matter; for, as Dr. Chevers tells us, the association of pericarditis and endocarditis with acute rheumatism is very rare in India, although articular rheumatism is very prevalent there.

The causes of *non-rheumatic* pericarditis are of two kinds, local and constitutional. The chief local causes are, cancerous diseases, arising within the sac, or, as much more frequently happens, spreading into it from some of the parts around the pericardium; tuberculosis, pleuritis, and peritonitis, by acting as local sources of irritation; abscesses of neighbouring organs or parts, and hydatid cysts, by opening into and discharging their contents into the sac.

The chief constitutional causes, or, to speak more correctly, the diseases in connexion with which the non-rheumatic pericarditis is observed, are diseases of the kidney, pleurisy, pneumonia, pyæmia, scarlatina, peritonitis, and variola.

Bright's disease of the kidney has been especially referred to by authors as a very frequent exciter of the inflammation. My own experience does not at all agree with this conclusion. Out of a great number of cases of kidney diseases which have come under my observation in the pathological department of St. Mary's Hospital, I have met but with very few where there existed any recent signs of pericardial inflammation. The same results have been arrived at in Edinburgh, where kidney disease is very

common. Professor Bennett (*Principles and Practice of Medicine*) tells us, "that in none of his cases of pericarditis has there been a complication with Bright's disease." Dr. Christison also says, "that pericarditis is rarely seen among the sequelæ of kidney disease." Frerichs also (*Die Brightische Nierenkrankheit*, p. 120) found inflammatory exudations in the pericardium in only thirteen out of two hundred and ninety-two cases of Bright's disease.

The following are the distinctive characters which appertain to these two classes of pericarditis :—

Rheumatic pericarditis occurs at a comparatively early period of life. The average age of sixty-one of Dr. Ormerod's patients was twenty-one. The subjects of the disease are, for the most part, of (apparently) healthy constitution. As regards sex, women appear as often as—perhaps somewhat more often than—men, to be affected by it. The symptoms of the rheumatic pericarditis are prominent and well marked; the disease, except in rare instances, being associated with, and in fact preceded by, rheumatic affections of the joints. The local pericardial inflammation, moreover, frequently reacts with violence on the system; and in such case, when death ensues, it is to the immediate consequences of the pericarditis—to the action of the inflammation on the heart itself—not to the *ordinary* effects of rheumatism, that the fatal result is to be ascribed. The heart is overwhelmed and paralysed by the local action of the inflammation

upon it. But though the symptoms of rheumatic pericarditis are usually severe, the disease, when uncomplicated with other cardiac diseases and pulmonary affections, rarely destroys life by its direct and immediate effects. A first attack of rheumatic pericarditis is, indeed, very rarely fatal.

Many observations lead me to suspect that there is an hereditary tendency, or family predisposition, to rheumatic pericarditis in the young; though, for obvious reasons, it is very difficult to arrive at any positive conclusions on this point. I have, for instance, remarked striking instances of the existence of cardiac disease in the children of parents who have themselves suffered from rheumatism and heart disease. The following are examples of the kind which have fallen under my notice :—

In one family, the father and two children had disease of the heart. The father had suffered from several severe rheumatic attacks before the birth of these children, and there was a loud cardiac bruit constantly audible over his heart. In the children, about fourteen and fifteen years of age, the heart was so impaired as to produce the severest symptoms of cardiac disease. One of them I found had suffered from pains in the ankles; but, as far as I could learn, the other had never suffered from any external symptom of rheumatism—her first complaints of illness having been short breath and dropsical swellings, the products of the cardiac disease.

In another family, the father had for years been a martyr to rheumatism; and his child, about two years and a half old, had organic disease of the heart.

Another child, ten years old, had had rheumatism, and when I saw her was suffering from mitral valvular disease. Her mother had been ailing twenty years, and was, I found, suffering from organic disease of the aortic valves.

Moreover, it is not at all uncommon to see two, and even three, children in one family attacked with rheumatic pericarditis; but then it may be fairly suggested, in such cases, that the children are all exposed to the same exciting influences of disease, and that, therefore, what has produced the affection in one may also have excited it in the other.

Non-rheumatic pericarditis, on the other hand, occurs at a later period of life than the rheumatic kind. Forty-two was the average age of persons affected by it in twenty-four cases reported by Dr. Ormerod. The subjects of the disease were, with a single exception, all of the male sex. The inflammation does not so much attack the healthy, as those whose constitutions are originally weak, or have been debilitated through ill habits of living. It is far more serious than rheumatic pericarditis in its immediate consequences.

Dr. Ormerod informs us, that 91·6 per cent. of his non-rheumatic cases were fatal; whilst of the rheu-

matic sort, 18 per cent. only were fatal. The local symptoms, again, of non-rheumatic pericarditis are at times very obscure—often, indeed, so obscure as not to direct the observer by any local signs to the seat of the disease; and thus it not unfrequently happens, that the existence of the pericarditis is discovered only after death. As corroborative of this fact, it may be observed, that the local inflammation does not appear to exercise, by reaction (as happens in the case of rheumatic pericarditis), any particular influence over the patient's general condition, so as to appear in any direct way a cause of death.

It is not, therefore, by the violence or the immediate effects of the local inflammation, that life is destroyed in cases of non-rheumatic pericarditis. Death rather results from the other effects of that particular disease which has, in fact, preceded, and given rise to, the pericarditis. Thus, for example, when pericarditis appears in the course of kidney disease, it usually supervenes at an advanced period of the renal disorder; but kidney disease (of the character here inferred) is, as we well know, for other reasons fatal in its consequence, and quite independently of its action upon the heart.

Thus, then, in *non-rheumatic pericarditis*, the local inflammation plays a subordinate part. Its share, as a disturber of the system, is but small; so that it is, in fact, usually little more than a local sign (unimportant in its immediate effects) of a serious general

disorder. In *rheumatic pericarditis*, on the other hand, the pericardial inflammation must be considered as the all-important fact; its consequences fall at once, and with violence, upon the heart, both impeding mechanically, and otherwise seriously deteriorating, its powers.

CHAPTER III.

GENERAL SYMPTOMS OF PERICARDITIS.

PULSE. — RESPIRATION. — PAIN. — CEREBRAL SYMPTOMS. — DYSPHAGIA.—RELATION OF PERICARDITIS TO RHEUMATIC ARTHRITIS. —PROGNOSIS OF PERICARDITIS.

THE general symptoms attending acute pericarditis vary considerably. They are, for the most part, merged in the symptoms of the particular disease with which the pericarditis may happen to be associated. The pericarditis of rheumatic fever, indeed, often supervenes in the progress of the fever without, in any degree, adding to or altering the general symptoms attending it. Sometimes local pain, and a feeling of oppression about the præcordial region, indicate that inflammation has fallen upon the heart; but every stethoscopist is aware, that, as a general rule, it is by the physical signs—the friction sounds—that we gain the earliest intimation of the existence of the pericarditis. This fact shows how important it is ever carefully to watch for these signs during the progress of all diseases in which, as experience teaches us, pericarditis may possibly arise.

Moreover, in severe cases of pericarditis, inflamma-

tion of other parts and organs frequently coexist,—endocarditis, pleuritis, and pneumonia, for example,—and necessarily modify and complicate the character of the symptoms. Hence, there are no general symptoms which can be considered as special indicators of the existence of pericarditis. In advanced periods of the disease where the inflammation is severe, and its exudations are considerable, general as well as local indications of impediments to the respiratory and circulating organs arise.

The *pulse*, as a sign of pericarditis, gives us little sure information. In the early stages of rheumatic pericarditis, we meet with the full, hard, and frequent pulse of acute rheumatism. But as the pericarditis advances, and when the effusion is great, the pulse becomes weak, fluttering, and irregular, indicating an oppressed and enfeebled condition of the heart.

The Respiration.—Several causes tend, directly and indirectly, to quicken the respiratory movements, and impede the respiration in pericarditis; and these causes act conjointly or separately. The due expansion of the thorax may be prevented by the pain to which its movements give rise; and the free return of the blood from the lungs be impeded by the pericardial effusion, which restrains and weakens the action of the heart, and compresses the auricles, as already explained. The motion of the diaphragm, the pleuritic surface of which is often involved in the inflammation, is also sometimes impaired, in conse-

quence of the pain which it occasions. Pleurisy and pneumonia, moreover, are frequently associated with acute pericarditis in its advanced stage; and when present, they also necessarily interfere with, and embarrass, the respiration; and more or less so according to their degree and extent. And again, where the pericardial effusion is considerable, it mechanically compresses the lungs, and particularly the left lung.

In consequence of these disturbances of the respiration, we find that the respiratory movement of the upper part of the chest is sometimes increased, and that of the epigastrium arrested. The expansion of the ribs over the heart and left side of the chest is also partially arrested, particularly when the costal pleura participates in the inflammation.

Hence the obstruction of the respiration which results from the pericarditis depends upon different circumstances:—upon the amount of pericarditic and pleuritic effusions; upon the degree of pain; upon the condition of the muscular structure of the heart; upon the presence or absence of pneumonia and pleurisy; and upon the extent and degree of these different affections.

Pain is, of all the symptoms of pericarditis, the most inconstant, and the least trustworthy. When present, it is felt chiefly about the præcordia and at the scrobiculus cordis, being increased on deep breathing, and by pressure—particularly by pressure at the epigastrium; it is increased also by motion.

Sometimes it reaches to the shoulder and down the left arm.

The pain attending pericarditis is of a twofold nature. There is the distress, the anguish, and the stifling feeling—the modified angina—which result from the oppression of the heart—its congestion; and there is the sharp, acute, and cutting pain, which probably, in the greater number of cases, depends upon the accompanying pleurisy.

It is doubtful, indeed, whether inflammation of the pericardium alone will produce this latter kind of pain. When the pain is absent, as it frequently is in slight cases of pericarditis, it seems to me probable, that there is also an absence of pleurisy. In some cases the præcordial pain, though actually present, may not be noted by the patient, on account of the severity of the accompanying arthritis.

Cerebral Symptoms.—Symptoms arising during the course of pericarditis indicative of disturbance of the nervous centres, are of very serious import, and for the most part precursors of a fatal termination of the disease. In young persons they present themselves under the form of choreal symptoms. Sudden movements of the body, twitchings of the limbs, throwing about of the head, great restlessness, delirium, distortion of the features, startings in the sleep, tetanic spasms, are the usual manifestations of these disturbances of the nervous centres. "Wild delirium, epileptic or tetanic convulsions, chorea, coma, fatuity,

are the greatest and the rarest of these symptoms; and mutterings, reveries, transitions from torpor to excitement, subsultus, are the least and most frequent, but they are all akin one to another. The least may mount up to the greatest, and the greatest run down to the least." (Latham.)

The old doctrine of metastasis gives no explanation of these symptoms. It is possible, as has been suggested, that the poisonous agent in the blood may act directly upon the nervous centres; or that the excitement may arise in a secondary or reflex manner, as a consequence of the local disease. But it seems more in accordance with modern pathology to consider that these nervous derangements result from profound alterations in the condition of the blood, which have been produced either by the disease, or by the treatment adopted for its cure. I have already observed, that since venesection and mercury have ceased to be considered as necessary remedies in pericarditis, the nervous symptoms alluded to seem to be less frequently met with by practitioners. The furious delirium which sometimes arises in pericarditis may be regarded in most cases as the consequence of exhaustion of the nervous power. It is in the advanced stages of the disease, indeed, that these nervous symptoms supervene.

The following case, which lately came under my observation, is a good illustration of this condition of disease in its typical form:—A. B., a painter, aged

thirty-seven, had for several weeks suffered from great dyspnœa, in connexion with pericardial disease. Three days before his death, he became delirious, and for the last two days of his life was furiously maniacal. His brain was found to be perfectly healthy; but a large amount of serous, subarachnoid effusion surrounded it. The pericardium contained three pints and a quarter of fluid, and stretched twelve inches across the chest. The heart was thickly covered with freshly deposited villous fibrin. The lungs were compressed backwards towards the spine, but were healthy.

We may fairly conclude, that here the mechanical pressure of the fluid upon the heart, and upon the lungs, was the chief immediate promoting cause of death; and that the delirium was the consequence of exhaustion, the patient having taken very little food during his lengthened suffering.

Difficulty of swallowing is occasionally observed when the pericarditic effusion is considerable; the difficulty being increased when the patient lies recumbent. It depends, we may surmise, upon the pressure of the fluid in the pericardium upon the œsophagus, whereby the passage of the food into the stomach is mechanically impeded. The same pressure has been thought, also, in rare cases, to have acted upon the trachea, and so to have interfered with the respiration; this I have never observed.

The condition of the skin depends, in great part,

upon the nature of the pericarditis, whether rheumatic or not. When the disease is of rheumatic origin, the state of the skin, as well as many of the general symptoms, are those peculiar to acute rheumatism.

In rare cases of rheumatic pericarditis, the inflammation of the pericardium has been known to precede the affection of the joints; sometimes it comes on when the arthritic affections have already ceased to exist; but much more frequently the pericardial inflammation supervenes during their progress.

It has not yet been clearly ascertained, whether the severity of the articular bears any relation to that of the pericardial inflammation. "Sometimes we find the one affection is violent, and the other absent or slight; and then, again, both the rheumatism and the pericarditis are violent, and their symptoms increase and decrease in force together. The fact is, that pericarditis may be looked for whether the acute rheumatism be severe or mild,—whether its seat be fixed or shifting, and at the beginning, as well as during the progress of the disease." (Latham.)

Such seems the experience of physicians in London; but it does not correspond with that of Dr. Stokes, in Dublin, who says: "The liability to all forms of carditis in rheumatism is in proportion to the severity and obstinacy of the fever." The question is one which is manifestly not easy of solution. In accordance, however, with what I have already

said in speaking of the causes of pericarditis, it is certain, that the mildest form of rheumatic fever in the young is more likely to be attended with cardiac affections, than the severest form of the fever in persons advanced in life.

Such are the most prominent of the symptoms attending rheumatic pericarditis. It is unnecessary for me to indicate especially the signs and symptoms of *non-rheumatic pericarditis*. The general symptoms which accompany this form of the inflammation are those of the particular disorder, in conjunction with which the pericarditis exists.

Prognosis of Pericarditis.—Rheumatic pericarditis usually runs a rapid course, and the subject of it, in a first attack at least, almost invariably recovers from its immediate effects. Judging, indeed, from the past history of this inflammation, we may infer, that since the use of large bleedings and mercurial salivation have been abandoned in its cure, the disease has become much less fatal.

Unfortunately, however, rheumatic pericarditis rarely fails to leave behind it, and especially in the young, marks of permanent mischief done to the heart—pericardial adhesions, disorganization of its muscular structure, and above all injury of its valves, caused by the endocarditis, which so very frequently accompanies pericarditis. It is impossible, therefore, to form a favourable prognosis of the health of a person who has been once attacked with rheumatic

pericarditis; for the heart, in such cases, appears especially predisposed to a repetition of the attack. Rare, indeed, are the cases in which the heart once attacked escapes permanent injury; rarer still those in which it remains ever after free from a repetition of the attack. Sometimes the injury is too slight to occasion symptoms such as may warn the patient of its presence, but is still marked enough to declare itself to the ear of the physician. Sometimes, again, when the injury has been more severe, the patient is conscious in himself that he is not the man he was before the attack; his breathing is shorter and quicker; he has an occasional cough; he is not able to undergo the exertion which he was once equal to; occasional palpitations also trouble him, especially if he walk fast or run. In short, he now bears about him, in a more or less marked form, the symptoms of heart disease. In all such cases, when positive signs of injury done to the heart remain—irregular action, or abnormal sounds—the patient must ever be the subject of watchful care. To him, if to any one, the motto of Corvisart is truly appropriate—*Adhæret lateri lethalis arundo.* A second attack of inflammation will, we must fear, still further disable the heart's textures; or gradually, and in a chronic way, a slow process of disease—degeneration—may affect the already imperfect valve, or the injured muscular structure; and thus the imperfection will be increased, so as at length to manifest

itself in symptoms to the patient, as well as in signs to the physician's ear.

Happily, there is no class of chronic maladies in which the medical art is of more avail in the relief of symptoms, and by its prophylactic measures in warding off further attacks of the inflammation, than in this, provided only the subjects of them are willing to submit to treatment, and are, by social position, enabled to bestow the necessary attention to their bodily condition. Under such favouring circumstances, it is really surprising how life may be long preserved, even when the structural disorganization of the heart is very considerable. But among the poor, those who are thus affected have but small chances of prolonged life. They are still exposed to the causes which are the original sources of their malady; and their very exertions to gain the means of living increase the severity of the symptoms which are destroying them. There are no cases met with in hospital practice whose early progress is more sad to watch than these. Removed from the immediate provoking causes of their sufferings, and subjected to proper hygienic rules, the improved change in the bodily condition of such patients is often surprising. The dropsical symptoms, the difficult breathing, the præcordial pain, the palpitations, &c., quietly disappear, and the patients after a time leave the hospital with the belief that they are cured of their disease. Then once more they return, as

they must, to their labours; and once more these labours, and the other attendant circumstances of their condition, provoke the rapid recurrence of the evils which they vainly hoped they had for ever left behind them when they quitted the hospital. For such persons prognosis has nothing favourable to offer.

CHAPTER IV.

AUSCULTATORY SIGNS OF PERICARDITIS.

PERICARDIAL FRICTION-SOUND.—DIFFERENCE BETWEEN FRICTION-SOUNDS AND ENDOCARDIAL MURMURS.—PERCUSSION SIGNS.—PERICARDIAL ADHESIONS.

We have seen that the general symptoms attending pericarditis—whether rheumatic or non-rheumatic—do not enable us to diagnose with precision the existence of the disease. In *non-rheumatic pericarditis*, indeed, the inflammation is, for the most part, of a latent kind, being insidious both in its origin and in its progress, and, from an absence of all local symptoms of its presence, not unfrequently entirely escapes observation during life.

And even in *rheumatic pericarditis*, where local symptoms of the inflammation—more or less pain, and a feeling of oppression about the præcordia—are usually present, we obtain the earliest certain information of its existence solely through the aid of the stethoscope.

The physical characteristics of pericarditis are:—double or single friction-sound heard over the præcordial region, and increase of the normal dulness heard on percussion there.

Friction-Sound.—A double or single friction-sound heard over the heart, synchronous with its movements, and confined to the præcordial region, is the surest sign we possess of the presence of pericarditis. Fever, præcordial pain or tenderness, and all the other general symptoms of pericarditis, may exist; yet, until this friction-sound be heard, we cannot with certainty affirm that the pericardium is attacked. The friction-sound appears at a very early stage of the inflammation; and the smallest amount of lymph exuded on the surfaces of the pericardial membrane will, I believe, give occasion to it, provided no fluid come between them.* It may remain audible through the whole period of the existence of the inflammation; or it may be heard at the commencement, rapidly pass away, and appear once again when absorption of the fluid part of the exudation has taken place. The friction-sound usually accompanies both the systole and the diastole of the heart; occasionally it is heard during the systole only; in some very rare cases, only during the diastole.

The loudness and character of the friction-sound are modified by several circumstances. Thus, the intensity of the sound depends not only upon the roughness of the surface of the fibrinous deposit, but

* I once distinctly heard pleuritic friction-sounds in a lad, the subject of pyæmia, a few hours before his death. The very thin layer of lymph—however readily seen when looked for—which occasioned the sounds, would, I believe, have been passed over unnoticed, except for the fact of the friction having been heard during life.

also upon the area of roughened surfaces which rub upon each other; upon the force of the heart's action; and upon the amount of fluid effused into the sac. Its varieties are only degrees of the same sound, embracing both the gentlest rubbing and rough, creaking sounds. We gain only confusion by giving these sounds a variety of names. The conditions requisite, therefore, for the production of the friction-sound are: the deposition of solid or semi-solid matters on the surface of the membrane; the contact of its opposing surfaces; and a certain degree of force in the action of the heart. Serous, hæmorrhagic, or purulent effusions, will not of themselves occasion a friction-sound, nor will the sound arise when the heart's action is very feeble, nor whenever such an amount of effusion is present as suffices to prevent the surfaces coming in contact, and so rubbing on each other—a condition, indeed, which very rarely obtains in a complete way.

The friction-sound terminates, if the patient recover, either in adhesion of the pericardial surfaces, or in the resolution of the inflammation; and, probably, sometimes in the formation of "white spots."

Difference between Endocardial Murmurs and Pericardial Friction-Sounds.—It is, in certain rare cases, difficult, if not impossible, to distinguish the pericardial friction-sound from an endocardial murmur. A great authority in stethoscopy asserts, indeed, that there is no kind of endocardial murmur (with the

exception of the whistling murmur) which may not resemble a pericardial friction-sound. In such cases we must judge of the nature of the sound by the period of its occurrence, and by the presence or absence of co-existing signs of endocardial disease. The friction-sound is generally double; it does not coincide exactly with the heart's sounds; it commences rather *before* the systolic sound is heard; and, as a rule, it is not audible in the direction of the current of the blood flowing along the vessels out of the heart. The friction-sounds, moreover, do not replace the heart's natural sounds, except when they are so loud as to overpower them; their duration is variable and limited; they change their seat; they vanish and reappear; they are generally confined to the præcordial region; and they vary in force and situation from day to day. Pressure of the stethoscope, also, as Dr. Sibson has shown, increases the intensity of the pericardial friction-sound in certain cases; but does not, he says, affect an endocardial murmur.

These differential signs, however, must be accepted with a certain degree of caution, and for the following reasons:—An endocardial murmur is very frequently present in pericarditis, and thus complicates the pericardial friction-sounds; the heart's action, again, may be so rapid, that the relation in time of the pericardial murmur to the heart's sounds cannot be determined; and a pericardial friction-sound may be heard beyond the præcordial region.

Even pressure by the stethoscope does not appear to be an infallible sign. Dr. Walshe states that he has heard a mitral murmur increased in force by it; and it seems probable that pressure over the site of the pulmonary artery may, in some cases, excite or increase a murmur there. The effects of pressure, again, can hardly be made to bear upon the pericardial surfaces in persons advanced in life, in whom the walls of the thorax are firm and unyielding. Neither can the pressure be exerted upon the affected parts, except when it can be made to bear directly against the roughened surfaces of the pericardium, and therefore not in cases where the friction-sounds proceed from the sides and back parts of the heart. A pericardial friction-sound may also be confounded with an exo-pericardial friction-sound, arising from the rubbing of the outer surface of the pericardium against some opposing surface of the pulmonary pleura, when this is covered with plastic exudation. The sounds—certain bronchial râles—resembling friction-sounds, which sometimes arise within the lungs, and likewise pleuritic friction-sounds, may be readily distinguished from pericardial friction-sounds by the circumstance of their cessation during the arrestment of the respiratory movements.

A blowing murmur—endocardial—is occasionally observed along the aorta, and over the left ventricle, in cases of pericarditis, unaccompanied by endocarditis, and in which, after death, no valvular lesion is

found to exist. The cause of such a murmur may be attributed either to pressure upon the aorta, produced by the exudation of lymph upon it; or to the loss of its elasticity, or some other alteration of its coats, caused by the inflammatory process; or to irregular action in the heart's muscular movements, involving those of its columnæ carneæ, whereby the function of the auriculo-ventricular valves is rendered temporarily incomplete. Or, again, when the murmur is persistent, it may possibly, I have thought, be ascribed to the pericardial adhesions; these being of such a character as to prevent the walls of the heart, and consequently the columnæ carneæ, from freely contracting, so that the mitral orifice is left partially unclosed during the heart's systole.

As a general rule, we find in health that about one to two inches* of the heart's surface is uncovered by the lungs. This uncovered surface may be rudely likened to a triangle. A line drawn perpendicularly down, and somewhat to the left of the centre of the sternum, from about the fourth to the sixth or seventh rib, defines the position of the anterior edge of the right lung, and constitutes the right side of the triangle; its left side is an irregular oblique line, corresponding to the anterior border of the left lung, and drawn from the fourth left sterno-costal articulation, downwards through the cartilages of the fifth

* Of course the extent of uncovered heart is greatly regulated by the condition of the lungs, whether inflated or empty.

and sixth ribs: these two lines meet above, directly beneath the level of the fourth rib; the base joins the sides above the cartilages of the sixth rib. The dull percussion is often ill-defined at the base of the triangle, because the left lobe of the liver in many persons lies in close contact (the diaphragm intervening) with the lower border of the heart.

Between the anterior surface of the heart and the thoracic walls only fat and intercellular tissue naturally intervene: hence, when dulness on percussion exists beyond the limits here indicated, we may suspect an abnormal condition of the parts beneath. The dulness in itself of course simply indicates that solid or fluid matters, containing no air, exist beneath the thoracic walls in those parts where a certain amount of air ought naturally to be present; the diagnostic value of the dulness must be decided by other signs.

The extent of dull percussion-sound varies during respiration and expiration: on a full and deep inspiration, when the lungs almost wholly cover the pericardium, the dulness is hardly perceptible; on a deep expiration it becomes proportionally increased.

Percussion Signs.—Effusion into the pericardium increases the ordinary degree of præcordial percussion dulness, but not always in proportion to the amount of fluid present. When the effusion is very considerable, the percussion may be dull over the greater

portion of the anterior surface of the left side of the thorax, and even beyond the right border of the sternum. Increased resistance, also, is in such case felt by the finger on percussion. The dulness arising from pericardial effusion is more marked than that which arises from hypertrophy and dilatation of the heart. Practised from day to day, percussion often enables us to follow the increase or diminution of the fluid in the pericardium. It does not, however, give us any accurate information as to the amount of effusion which has taken place. A small amount of pericardial effusion, for instance, may be associated with hypertrophy of the heart, with malignant disease, with enlargement of the left lobe of the liver, with aneurism of the aorta, with condensation of the lung,* and with pleuritic effusion; and consequently the extent of the præcordial dull percussion-sound may, in such cases, be increased out of proportion to the amount of pericardial effusion. Enlargement of the liver, also, particularly of its left lobe, occasionally pushes the heart upwards and to the left; encroaching on the præcordial region, and increasing much the præcordial dulness. On the other hand, where there is emphysema of the lungs, or consolidation of the posterior parts of the lungs, the anterior

* Pneumonic consolidation of the parts of the lungs which overlap and surround the pericardium, very frequently co-exists with pericarditis—being, apparently, excited by the contiguity of the pericarditis. Such consolidation will, of course, of itself occasion an increase of the præcordial dull percussion-sound.

parts being inordinately extended,* and consequently overlapping the heart to an unusual extent; or when the lungs are adherent to the thoracic walls in front of the pericardium; or where there is atrophy of the heart—the amount of effusion present may appear, on percussion, to be less than it really is. An inflated stomach, also, often modifies considerably the percussion-sound over the præcordial region.

The fluid effusion collects, in the first instance, about the base of the heart and the roots of the great vessels; and it is in their situation that the abnormal dulness is first observed: but as the quantity of effusion increases, the dulness reaches across to the right, or beyond the right border, of the sternum, upwards towards the clavicle, and towards the left lateral region—that is, in the direction of the heart's breadth; the heart, being heavier, sinks downwards and backwards in the pericardium when the patient lies recumbent; and its apex is pressed somewhat to the left of its natural position.

We gather from the above that increased præcordial dulness is, in itself, no certain sign of pericardial effusion; but, judged of by the light of other signs,

* I have observed cases of this nature after death, where the anterior border of the right lung, in consequence of the respiration being carried on by the anterior portions of the lungs, lay far over the distended pericardium, and imparted a clear sound to moderately forcible percussion over the sternum; and this, too, even when the distended pericardium reached beyond the right border of the sternum.

it becomes so. Pericardial effusion commences at the anterior, pleuritic effusion at the back and sides of the thorax. In hypertrophy of the heart, the pericardial dulness is not so well marked, nor is the resistance so complete as in pericardial effusion. And, besides this, hypertrophy has its special signs to distinguish it: in effusion the dulness commences suddenly, and increases most markedly in an upward direction; in hypertrophy, downward and towards the left side, or in the direction of that particular part of the heart which is enlarged.

Together with the friction-sound and other signs of pericardial effusion, a *fremitus* is said to be sometimes felt over the præcordial region when the hand is laid thereon. In young subjects, when the elasticity of the ribs and their cartilages permit of it, a bulging of the præcordial region is sometimes observed where the pericardial effusion is considerable.

The Heart's Sounds, Impulse, &c.—At the onset of the inflammation the heart's sounds are generally louder, and its impulse stronger and more extensively felt over the præcordia, than natural. But as the inflammation advances, and when exudation gathers in the pericardium, the movements of the organ become feebler; its impulse irregular and trembling; and its sounds at last weak and altered in character, or superseded by morbid sounds. When a considerable quantity of effusion has taken place into the

pericardium, and the patient lies recumbent, the heart naturally gravitates to the back of the pericardium; and, under such circumstances, the natural sounds and the impulse of the heart may become very weak, so as sometimes to be almost imperceptible.

Pericardial Adhesions.—Adhesions of the pericardium offer no positive signs by which we can with certainty recognise their existence during life; but when we have observed the progress of the pericardial inflammation from its commencement, and the gradual departure of the friction-sound, and of the other symptoms of pericarditis, in any particular case where the disease has been extensive, we may anticipate, with tolerable certainty, that adhesions have taken place.

Skoda has pointed out signs which he considers indicative of pericardial adhesions; and these I will shortly mention. It must be premised, however, that where these signs exist, union is supposed to have taken place, not only between the pericardial surfaces, but also between the external parts of the pericardium, the neighbouring pleural surfaces, and the walls of the thorax.

His diagnosis is based upon the signs which show that the heart's apex is drawn *upwards and to the right* during the systole, being prevented from moving downwards and to the left.

The heart's apex gives no systolic beat; it is

either not to be felt, or seems to cause a shock during the diastole.

During the systole, depressions are visible in the intercostal space corresponding to the apex, and frequently in one or more spaces above it.

Systolic retraction of the left intercostal spaces does not of itself enable us to diagnose adherent pericardium ; it must also be shown that, simultaneously with the retraction, the heart's apex is nowhere urged against the thoracic walls.

Dr. Law, in the *Dublin Quarterly Review*, vol. xxii., p. 81, gives as a sign of adherent pericardium—"the persistence of the same extent of dulness to percussion in the præcordial region, no matter what position the individual may assume."

The only one of these signs which I have been able to verify, is the permanence of dull percussion over a given part, during inspiration as well as expiration ; but this fact necessarily indicates also exo-pericardial adhesions.

CHAPTER V.

TREATMENT OF PERICARDITIS.

BLEEDING.—MERCURY.—POTASH.—OPIUM.—GENERAL AND LOCAL TREATMENT.

We have already seen that a marked distinction exists between the pathological characters and the symptoms of the rheumatic and non-rheumatic forms of pericarditis; and we shall now find that a similar distinction is to be made in their treatment. The treatment of the *non-rheumatic pericarditis* is the treatment simply of that particular general affection with which it happens to be associated—uræmia, scarlatina, tuberculosis, pyæmia, erysipelas, &c. In non-rheumatic pericarditis, the local inflammation—the pericarditis—as a rule, demands no special consideration, as an object of treatment.

On the other hand, in rheumatic pericarditis we have to direct our remedies both against the local inflammation, as well as against the general specific disease, which excites the inflammation.

We have to neutralise, if possible, the action of the specific disease; and we have at the same time to assist the heart in struggling through and against

the inflammation and its consequences. It is, therefore, to the acute form of the disease, as especially typified in rheumatic pericarditis, that the following observations on its treatment apply.

Bleeding by venesection in the treatment of pericarditis has been highly extolled; but at the present moment its practice is rarely resorted to. It is admitted by common consent, that its value, as a remedial agent, has been overstated by Bouillaud, and other extreme admirers of the practice.

A better pathology and a truer method of observation have forced upon the physician, during the last few years, a conviction of the inutility, and sometimes injurious effects of large venesections upon the progress of internal inflammations; and even they, happily few, who still follow the practice of former days, in the treatment of acute internal inflammations, admit that it is only during the very early periods of the pericardial inflammation, and in patients of strong and robust constitutions, that venesection is of service. That a moderate bleeding may be practised with impunity, *under such conditions*, there is little doubt; and that it often is of much benefit in giving temporary relief to the sufferings of the patient, and in relieving the congestion of the heart and lungs, is certainly true; but that such a bleeding has *any other beneficial effect or any direct influence over the progress of the inflammation has yet to be proved.*

In considering the question of the value of venesection in pericarditis, we must remember:—That every inflammation is a disease of weakness, a condition which, in reference to health, is an asthenic condition; That heat, redness, and increased vascularity are no more signs of a sthenic circulation, than is the hyperæsthesia of a paralysed limb a sign of increased vigour of its nerve force; That rheumatic pericarditis occurs exceptionally only in persons of robust constitution; That an organ is involved in this inflammation, the constant performance of whose functions is indispensable to life; And that one of the most immediate effects of the inflammation and its products is to induce a paralysed condition of the muscular structure of the heart; That in pericarditis, the reaction of depression, consequent upon the excitement, is great, and sets in early; and that it is in those cases in which the inflammation appears most violent at the onset, that we are most cautiously to watch for and to expect the greatest amount of subsequent depression; That bleeding, moreover, will not arrest the exudation, but, on the contrary, in certain states of the body, will hasten and increase the amount of it; and that, inasmuch as endocarditis is very commonly associated with the pericarditis, bleeding, by promoting the tendency in the blood to the deposition of its fibrinous particles, will increase the danger of permanent injury to which the valves are exposed, through the deposition of

fibrine upon them ; And that venesection, as we well know, increases the amount of fibrine in the blood, and diminishes that of the red globules—the increase of the one and the diminution of the other being both indicative of weakness.

Another special danger has been strongly pointed out by Dr. Todd. (*Renal Diseases*, p. 412.) He says :

"An active antiphlogistic treatment creates asthenia ; asthenia gives to both rheumatic fever and gout what I may call *the shifting character*, which in both diseases is most perilous. When you find this shifting tendency, depend upon it that the asthenic condition of the patient is that which demands your earliest attention." "In this case of acute rheumatism," he says again at p. 12, "the loss of large quantities of blood from hæmaturia at an early period of the disease, has not sufficed to keep off a severe attack of pericarditis . . . . nor has it saved the patient from swollen and exquisitely painful joints. On the contrary, the articular, as well as the cardiac symptoms, have been much less tractable than usual."

Experience has also shown us, that venesection has no *directly* beneficial influence over pericarditis ; and that large bleedings are prejudicial, and therefore inadmissible in this disease. Nevertheless, that small bleedings are often of very great service *in relieving the congestions of the heart and lungs*, which so often arise as consequences of and coincidently with the

pericarditis, is, I think, an undoubted fact. I have elsewhere spoken of this particular mode of action of venesection; and if the conclusions* there arrived at are, as I firmly believe, correct, it necessarily follows: that moderate venesection, practised for the object indicated, viz. to relieve the congestion of the heart and lungs, may be often resorted to with great benefit to the patient.

In all stages of diseases of the heart in which congestion of the organ occurs, such venesection—duly adapted to the individual case—is of great service. I consider that I have even seen life preserved by timely abstraction of blood in cases of chronic valvular diseases of the heart, where the organ was so overwhelmed and labouring as to render death imminent. (See Appendix I.)

Local abstraction of Blood.—Whenever pain on pressure, or on inspiration, exists about the præcordial region, during the early periods of the pericarditis, there can be no doubt as to the propriety of

* 1. Venesection has no *directly* beneficial influence over the course of inflammations, either external or internal.

2. But venesection is at times of great service, *indirectly*, in the course of certain internal inflammations, and in the course of certain internal chronic diseases, and of all disorders which occasion congestion and oppression of the heart.

3. The use of the venesection is in all cases alike. It acts by relieving the cardiac oppression, and the coincident pulmonary congestion; it neither arrests nor modifies, beneficially, inflammation. (*Remarks on the Uses of Bleeding in Diseases.*—British Medical Journal, 1858.)

taking blood, by bleeding or cupping, from that part. The blood is, in such case, drawn *directly* from the inflamed pleura (through the intercostal arteries) which always co-exists with severe pericarditis, and, probably, from the inflamed pericardium, through the anterior mammary artery.*

It has been thought that leeches applied to the inflamed joints may favourably influence the pericardial inflammation; but of this there is no proof. I have, however, so often seen great relief given by the application of one or two leeches to the inflamed joints when the arthritic pain was severe, that I cannot doubt the propriety of their use under such circumstances.

* There is, as I have elsewhere endeavoured to show, a marked distinction to be drawn between the effects of venesection upon inflammation and the local abstraction of blood from an inflamed part. Local abstraction of blood materially influences the inflammation, reducing the most characteristic of its phenomena. But local abstraction of blood can only modify, in this way, the inflammation of *internal* parts, when there is a direct vascular connexion between the part inflamed and the part from whence the blood is taken.

The distribution of the blood-vessels enables us to explain the reason of the beneficial influence of the local bleeding in pericarditis. The internal mammary artery, which, in part, supplies the skin, &c., over the left side of the sternum, sends a branch directly to the pericardium. The intercostal arteries, also, which supply the costal pleura, are in part distributed to the skin over the præcordial region. Consequently, local bleeding acts directly, both upon the inflamed pericardium and the inflamed pleura, by drawing blood or diverting the current of blood, from the part.

Mercury.—The induction of the specific effects of mercury was once considered essential in the cure of acute pericarditis, but the practice is now generally abandoned. Dr. Taylor's observations first demonstrated its inutility. He showed, that of forty well observed cases, only four improved after mercurial action was established; and there was no proof that in these four the improvement was other than a simple coincidence. Subsequent experience has fully confirmed Dr. Taylor's opinion. The practice never seems to have been much resorted to on the Continent; and in Germany is untried and unknown. Gendrin's opinion, that mercury is completely useless in pericarditis is the general opinion of his countrymen.

Mercurial preparations, however, are undoubtedly of great service when used to regulate the secretions, which are always more or less disturbed during the progress of the inflammatory disorder.

Salts of Potash.—Observation has proved that in the treatment of acute rheumatism, there is no remedy which has such a powerfully beneficial influence over the disease as the salts of potash, when administered in sufficiently large doses. The proof of this assertion is to be found in the results of our daily practice; and in the concurrent—I may say, almost universal—testimony of competent observers in different countries.* When one to two scruples,

* The following are a few of the authorities who may be

according to the age of the patient, of the bicarbonate of potass, much diluted, are given every second hour, in cases of acute rheumatism, a marked amelioration of the symptoms is generally—though, I admit, not invariably—observed to follow its administration in the course of about twenty-four or thirty-six hours,—in fact, whenever the urine becomes alkaline.

My own observation fully bears out the following statement of Dr. Garrod, and the authority of many competent observers might be cited in support of it. "Besides the influence on the duration of the articular affection which has been alluded to, I cannot help thinking that an effect is likewise produced on the cardiac disease, to a very considerable and important extent. In no case did the affection of the heart ensue after the patient had been more than forty-eight hours under the influence of the medi-

referred to on this point:—Dr. Basham gives from one to three ounces of nitrate of potash in four pints of water during the twenty-four hours. (Med. Chir. Soc. Trans., 1848.) Gendrin also gives nitrate of potash. (Med. Gaz., 1848.) Dr. Golding Bird used the acetate of potash in doses of half an ounce in the twenty-four hours, much diluted. Dr. Swett administers one drachm of the tartrate of soda and potash every two or three hours, until the urine becomes alkaline. (New York Med. Times, 1854.) Dr. Garrod's method is to give "two scruples of the bicarbonate of potash every second hour, night and day, in a wineglassful of water, until the joint-affection has ceased for three or four days" (Med. Chir. Trans., 1855); to this excellent paper I would especially call the attention of those desiring fuller particulars in this matter.

cine; and it has appeared to me, that even when present on admission into the hospital, or coming on within a short period, its progress was powerfully checked by the treatment, and prevented from producing the terrible mischief which, when uncontrolled, it so frequently induces: this I should be inclined to ascribe to the altered condition of the blood, and especially of that portion giving rise to fibrinous deposits on the peri- or endocardium." (*Loc. cit.*)

Now, inasmuch as it is highly probable, that the *materies morbi* which produces the rheumatic arthritis is identical with that which produces the rheumatic pericarditis, and as the potash treatment admittedly has a beneficial influence over the arthritis, we may reasonably conclude, that the remedy will be also efficacious in preventing or arresting the progress of the pericarditic inflammation. Whether the potash acts by neutralising or eliminating the poisonous element, or whatever be its action, is a matter of secondary consideration.

Opium.—The pain which afflicts the patient during the progress of acute rheumatic pericarditis is sometimes very severe. In cases of this kind, it is necessary to tranquillise the nervous system. We must remember, that the shock of violent pain will occasionally destroy life; and that a lesser degree of it, unceasing and severe, will likewise kill, by wearing out and exhausting the vital powers of the patient.

I have already pointed out how apt the vital power of the heart is to fail in pericarditis, through the local influence of the inflammation on its muscular tissue; and therefore we have especial reason to subdue or remove this other exhausting influence—the pain. Administered to this end—to relieve pain, and to procure sleep—opium will be often found to act with all its magic charms. So striking, indeed, is the relief it thus affords, that the drug has been thought to possess a power beyond that of merely procuring rest and alleviating pain. A direct influence in arresting the pericarditic inflammation has been ascribed to the opium; but whether it has any such directly beneficial influence over the local inflammation has yet to be proved. The solution of the question is not easy; for physicians do not consider themselves justified in using it alone.

Whenever I have seen it given, or have used it myself to produce, and keep up, a quiescent and soporific state, the potash treatment has been always simultaneously followed out.

The quantity of opium administered must be measured by the degree of pain suffered: and the large doses of it which even young children will take with impunity in these cases, is surprising. I have seen this remedy very extensively used, for this purpose, in St. Mary's Hospital, by my colleague, Dr. Sibson. The opium he gives to an adult, in doses of one grain, mixed with half a grain of compound colo-

cynth pill, every two, three, or four hours, sometimes every hour, for many hours together, until the main object of its administration—relief to the patient's sufferings—is obtained. It is very rarely that any of what are considered the ill effects of opium are observed to follow this use of it. It is not found to "lock up" the secretions, or to contract the pupil. It acts favourably on the skin, as an "eliminator;" it takes the patient pleasantly—"jucundé"—through his disease; and is often followed by a rapid convalescence.

Summary.—The *general* treatment, then, of rheumatic pericarditis is in the main—at least, during its first periods—the treatment of acute rheumatism. In the commencement of the inflammation, the *local abstraction* of blood, proportioned to the degree of pericardial pain and the strength of the patient, is of service. Should there be signs of co-existing pulmonary and cardiac congestions, small venesections should be resorted to for their relief. Warm fomentations or poultices should be constantly applied over the præcordial region—a method of bringing warmth to the inflamed part, and of giving relief to the patient, which is worthy of more attention than is generally given to it in this country. The beneficial effects of blisters have been doubted by some observers; but I have so often seen them followed by mitigation of the local pain, and apparently also by diminution of the pericardial effusion, that I do not hesitate to use

them, when the acute periods of the inflammation have passed away. They do not prevent the application of poultices or fomentations. Dr. Todd advises their use even in the acutest stages of the inflammation.

The secretions of the body should be duly attended to during the whole course of the pericarditis; for there is, we must consider, a poison to be eliminated from the system. The bowels should be regularly relieved, and the action of the kidneys maintained. The action of the skin, evidently the chief natural eliminator of the rheumatic poison, requires no promotion in this affection. Frequent purging must be avoided, for the partial exposure to cold, which is its necessary consequence, and the extreme pain and suffering which movement is apt to occasion in these cases (when the co-existing inflammation of the joints is severe), more than counterbalance, by the nervous excitement they produce, the good which the purging might be supposed otherwise to bring about, as a process of elimination.

In every case of pericarditis, whether rheumatic or non-rheumatic, and at all periods of the inflammation, the condition of the heart's powers should be carefully watched. Light and easily digestible nourishment—milk and broth—should be administered during the earliest periods of the inflammation; and stimuli, when the acute period has ceased, and the process of absorption has commenced, or

whenever the signs of enfeebled circulation begin to show themselves.

Of the treatment of the secondary disorders which result from the pericarditis through injury done to the heart's structures—of congestions of lungs, abdominal organs, and brain, &c.—I shall speak hereafter, under the head of valvular diseases.

The *treatment of non-rheumatic pericarditis* requires no special consideration. The treatment is mainly that of the particular disease with which it happens to be associated.

Paracentesis of the pericardium has been recommended and practised in certain cases where large collections of fluid have taken place in the pericardium, and have resisted the ordinary methods of treatment employed for promoting their absorption. The results of the operation in the limited number of cases hitherto recorded cannot be considered as satisfactory: but it must be admitted that no just conclusions as to its real value can be drawn from them, for they were cases in which the effusion was excited and maintained by the presence of tubercular or carcinomatous disease, in which the lungs and pleura were also more or less seriously damaged, and in which, therefore, a permanent cure was not to be expected.

The operation, as far as we can judge of it *à priori*, does not appear likely to be productive of any injurious consequences, if carefully performed; and the

only objection to its use in such cases seems to be, that it holds out no hope of permanent benefit. But there are cases, undoubtedly rare, in which I should not hesitate to recommend its performance—cases, for instance, in which a large quantity of fluid has been rapidly exuded into the pericardium, and, by its mechanical pressure upon the heart and lungs, seriously interferes with their actions. The simple puncture of the pericardium, inflamed and distended with fluid, can hardly be considered, under such circumstances, as even so important an operation as puncture of the pleura. It must be admitted, however, that in the acute and early periods of the pericarditis, the absolute necessity for the performance of the operation can with difficulty be shown. In one case in which I contemplated the performance of the operation, I found the patient, on the following day, so much recovered as to render its performance inadmissible.

Still I am inclined to think that the operation is called for in cases of the following kind :—A patient had been ill four or five weeks, and died at last furiously maniacal. Here, during life, extensive dull percussion had been observed across the front of the thorax. After death, I found the pericardium distended with three pints and a quarter of yellow lymph, and stretched twelve inches across the thorax : the pericardial surfaces were covered with false membrane. There was no other disease whatever to

account for death. The lungs were remarkably compressed upwards and backwards by the pericardial effusion, but were otherwise perfectly healthy.

The following case, recorded in the seventh volume of the *Pathological Transactions* by Dr. Barker, is also one in which we may fairly surmise that the operation would have been of service. Here the pericardium contained about four pints of pus, and the lungs were much compressed. There does not seem to have been any other disease. The man was young and healthy, and his prominent symptom was oppressed breathing. In fact, it would seem as though in this case death had been caused by the mechanical pressure of the purulent fluid on the heart and lungs :—

J. F., aged 26, a labourer, never very healthy, was attacked with pain of the left side of the chest three weeks ago. Breathing, 45 per minute. Front part of the left side of the chest larger than the right. Excessive dulness on percussion, reaching above to the lower margin of the left first rib ; below, to the lower margin of the thorax ; an inch to the left of the left nipple, and an inch to the right of the sternum. Elsewhere the chest was resonant. The heart's impulse nowhere perceptible ; its sounds feeble and distant. He died six days after admission into St. Thomas's Hospital.

The most appropriate spot for puncturing the pericardium is in the fourth left intercostal space, about three-quarters of an inch from the sternum, in order

to avoid the internal mammary artery. Iodine injections have been thrown into the pericardium by Aran and others, but for what purpose does not appear very clear.

I have not thought it necessary to say anything of what is called chronic, as distinguished from acute pericarditis. The term is now nearly obsolete. In both cases the inflammation and its consequences are alike; the difference being, that in the one case, the acute, the inflammation runs its usual course; whilst in the other, the chronic, the cause provoking the inflammation continues still in action, and therefore prevents the inflammation coming to its ordinary and, so to say, natural conclusion.

CHAPTER VI.

ENDOCARDITIS—PATHOLOGY.

ENDOCARDITIS.—VEGETATIONS, FIBRINOUS DEPOSITS, ETC., ON THE VALVES AND ENDOCARDIUM.—INJURY DONE TO THE VALVES BY THE INFLAMMATION.—CHRONIC VALVULAR DISEASES.—ATHEROMATOUS, CALCAREOUS, FIBRINOUS DEGENERATIONS.—SUDDEN RUPTURE OF VALVES.

By the term endocarditis is understood inflammation of the whole or of a part of the membrane which covers the valves and the internal surface of the heart. Included also under this head are all those morbid changes which the valvular structures undergo in connexion with acute diseases, or which are indirectly the consequences of endocardial inflammation.

Much obscurity still involves the pathological history of endocarditis; for now-a-days, happily, we have rarely an opportunity of studying the disease in its early stage. The remarkable researches and experiments of Dr. Richardson appear, however, to throw some light upon this subject; and I shall, therefore, state shortly what he has observed in cases

of endocarditis induced artificially by the injection of lactic acid into the peritoneum of dogs.

In considering this subject, it is necessary to keep in view the anatomical nature of the endocardial membrane, and the peculiar accidental circumstances which attend upon and influence its inflammation. This membrane is continuous with, and very similar in structure to, that which lines the arteries and veins. It consists of a stratum of fine fibres, bearing upon them a layer of very delicate epithelium. Blood-vessels ramify in the fibrous tissue, and it is from these that the pathological products of the inflammation are derived.*

The peculiar circumstances attending this inflammation are—the unceasing motion of the part inflamed, and the mechanical action of the blood, which is constantly flowing over and in contact with the inflamed surface, and with any exudations which may escape from it.

The deep red colour which the internal surface of the heart frequently presents after death, is, in almost all cases, the result simply of imbibition by the heart's tissues of the decomposed and dissolved colouring matter of the blood. The redness of congestion—the true inflammatory redness—is described by Rokitansky as constantly presenting a pale, rose-

* The idea that the internal membrane of the heart or arteries is nourished by the blood which flows over it, is quite contrary to all physiological facts.

red colour, the tint of it being subdued by the superficial layers of the endocardial membrane; it assumes a stripe-like, ramifying appearance, corresponding to that of the vessels, and has not the appearance of a general saturation of the tissues. Small ecchymoses are seen here and there in the inflamed part.

In the first stage of the inflammation, artificially produced by lactic acid, Dr. Richardson (*On the Coagulation of the Blood*, p. 371, *et seq.*) found the endocardial surface "intensely vascular, approaching in colour to bright vermilion; the membrane also had a soft, fleecy character, giving to it a velvety appearance." The valves were at the same time swollen and vascular, and clear lymph oozed from them when pricked. This lymph appears occasionally to have transuded through the membrane, and may have thus laid the foundation for fibrinous deposits from the blood, by causing its coagulation—an important observation, if further corroborated. "On the surface of the valve (mitral) there was a small fibrinous deposit, which adhered to its position by means of an underlayer of a white glutinous fluid, the like of which I found afterwards could be made to exude from the valve on puncturing it with a needle." Small beads were at the same time developed in the margin of the valves, and also yielded a clear, coagulable lymph when pricked, and then shrunk away.

In the second stage of the inflammation, the endo-

cardial surface became less vascular, and had a pale pink colour. The valves also were less red, but were still thickened with the now semi-solid exudation within them; and at points their tendinous chords were attached by loose adhesions to the endocardium. In the third stage, all redness disappeared; the exudation was firmer; and the valves, and their beaded edges, for a long time retained considerable firmness, and a pearly whiteness.

From these observations it would appear, that the ordinary products of inflammation are exuded both upon and within the endocardium. There are, therefore, two kinds of endocardial deposits to be considered:—The "beads," the granulations, and thickenings, which are produced solely by the inflammatory exudation thrown out beneath the unbroken endocardial surface, and therefore within the valves; and the "vegetations," &c., formed chiefly from the fibrin of the blood flowing through the heart, which is attracted to and deposited either upon the surface of the inflamed membrane, or upon the inflammatory matter that has exuded through it. These vegetations, therefore, are engrafted on the inflammatory deposit. It seems very probable, indeed, that a slight degree of roughness of the endocardial surface, resulting from the inflammatory action, may thus induce a deposition upon it of the fibrin of the blood; so that in this way the inflammation becomes, indirectly, a cause of the formation of fibrinous

coagula. And this is still more probable in diseases such as acute rheumatism, in which the blood appears to have a special tendency to separate it from its fibrinous constituents.*

These abnormal products, thus formed upon or exuded within the inflamed endocardium, present themselves to us under a variety of forms, differing much in size, number, and consistence. At one time they exist as minute granulations clustered together around the bases, or fringing the borders of the valves; sometimes they are attached, like polypoid bodies, to the valves, in an isolated form, varying in size from a pea to a hazel-nut. Then, again, they take the form of cauliflower excrescences, passing between and separating the valves, or fringing their torn and ulcerated edges. The fibrinous deposits are also frequently found fixed, not only on the valves, but also on the endocardium in their neighbourhood. They are mostly found fixed to that surface of the valves which is turned towards the current of the blood flowing through the heart. The

* Virchow asserts that these fibrinous formations are merely layers of fibrin separated from the current of blood which flows over the membrane. The only difference which exists, in his view, between endocarditis and endarteritis, is in the more acute course of the former, and in the tendency of it to the formation of papillary or warty excrescences. Both these facts he attributes to the greater vascularity and looseness of the cellular tissue which lies between the valves and beneath the inner layer of the endocardium.

different appearances which these granulations and vegetations, &c., present are readily explained, when we regard them as being formed, first, by the deposition of fibrin of the blood upon an inflamed surface: for whenever this surface is broken or roughened by inflammation, a deposition of the fibrin of the blood (as it would appear) readily takes place upon the broken part; and, secondly, as the results of inflammatory exudation, having the endocardial membrane still unbroken upon them.

The valves and their chords are not unfrequently ulcerated and ruptured, as a consequence of endocardial inflammation. The tendons of the papillary muscles, and the valves themselves, are also sometimes fused together, or with the neighbouring parts, so as to render the closure of the valves imperfect, or to diminish the aperture they surround. The aperture is also sometimes constricted by the deposit of inflammatory exudation in the tissues which form its circumference.

Endocarditis occurs more frequently, or at least its action is much more severe, in the left than it is in the right side of the heart.* The resolution of it

* I am inclined to think that endocarditis attacks the right side of the heart more frequently than is generally supposed; and that we do not notice the fact, because its effects are much less severe, and the pathological signs of it less well marked. In most cases, when the mitral valve is much diseased, the tricuspid will, I believe, be found also affected, although of course in a very much less degree.

is rarely perfect. Whenever we are able to diagnose the presence of endocarditis, we generally find that some appreciable consequences of the inflammation remain after the inflammatory symptoms have passed away. Under very favourable circumstances, it is possible that the inflammatory products may be entirely removed. The fluid exudation may be absorbed, and the fibrinous portion of them—the vegetations—formed out of coagula of the blood, be removed by the disintegrating action of the blood which flows over them. The fibrinous particles thus removed from their points of attachment, and carried along in the current of the blood into the general circulation, occasionally give rise to secondary disorders in the lungs, the brain, the kidneys, and other organs of the body, and sometimes appear to infect and alter the constitution of the blood itself.

The formation of abscess in the walls of the heart, as a result of endocarditis, is a most rare occurrence; when it exists, it may occasion rupture of the heart's walls.

Endocarditis is especially of interest, as being a cause of valvular diseases. Its chief effects, indeed, appear to fall upon the valvular apparatus; and when this has not been injured by the inflammation, and when the muscular tissue of the heart has not been damaged by it, the consequences of endocarditis are of minor importance.*

* It must not be forgotten, however, that the inflammation

Chronic Valvular Diseases.—Diseases of the valves of the heart, however, are not the products solely of inflammation. They are also, and perhaps in the majority of cases which occur at advanced periods of life, the consequences of a slow degeneration of the textures which form the valves. Chronic diseases of the valves are obscure in their origin, and slow and insidious in their progress. It is only in their advanced states, when they have so destroyed the integrity of the valve's structures as to occasion them imperfectly to perform their office, that they make their existence known to us during life by physical signs and symptoms. They present themselves to us under the form of abnormal depositions of fibrinous, atheromatous, and calcareous matters, beneath the endocardium, and within the valves. The quantity, and form, and nature, and stage of the deposit in each particular case, is the measure of the amount of injury done to the valve which it has pervaded. These peculiar degenerations of the textures of the heart and vascular system generally are, in one shape or other, so frequently observed in advanced life as to lead to the idea that they are modes of the natural decay of the tissue. When met with at

may be, and frequently is, the cause of a slow degeneration of the muscular tissue, through the damage it has done to its nutrition. This injury is less noticed than the valvular injury, because it appears less immediately connected with the inflammation.

early periods of life, they must be regarded as the consequences of perverted nutrition, resulting from unhealthy conditions of the system. There is reason to believe, for instance, that the fibrinous and atheromatous deposits in the valves and arteries are promoted by excessive indulgence in alcoholic fluids; and there can be no doubt that all habits and causes which tend to weaken and injure the system generally, assist in promoting any existing tendency to the deposit of these materials in the tissues. Moreover, it is well to remember, that true inflammatory exudations may also undergo these degenerative changes.*

Valvular diseases, as here considered, result from the destruction and disintegration of the tissues of the valves when invaded by abnormal—fibroid, fatty, and calcareous—matters. The natural properties of these tissues—their remarkable strength, firmness, and elasticity—are thereby diminished or destroyed; so that, softened and brittle and unfitted for their function, they tear, rupture, and yield under the pressure and distension to which they are of necessity continually subjected by the action of the heart. The deposits take place, for the most part, beneath and within the internal membrane of

* Under this head, I would particularly call attention to some very interesting researches of Mr. Canton, which certainly tend to upset the theory of atheromatous degeneration being a special attribute of old age.—(See *Lancet*, 1859-60.)

the valves of the heart and vessels, and consequently they cut off its nutritive supply, which, in the case of the arteries, is derived from the vasa vasorum, through the medium of the middle coat. Hence, also, arises a cause of ulcerations and destructions of parts. The deposit is at one time chiefly atheromatous, and soft; at another, by the accumulation of calcareous salts, the tissues become hardened—brittle plates of greater or less extent being thus formed in it, and sometimes so extensively as to convert the valve or the artery affected into semi-solid textures. Under such circumstances, when the internal membrane is broken or destroyed, the roughened borders and points of the calcareous plates become exposed to immediate contact with the blood; and hence the fibrin of this fluid not unfrequently becomes attracted to and deposited upon them, just as we found it was in the case of endocarditis. The blood, also, under such conditions, is apt to assist in the further destruction of the affected parts, being forced at each systole of the heart beneath the edges of the calcareous plates, and against the now weakened coats of the vessel. It is in this way that it usually distends the arterial coats in the formation of aneurisms, and aids in the rupture and ulceration of the valves of the heart.

We not unfrequently meet in practice with cases in which one of the aortic valves appears to have been suddenly ruptured in this way by violent mus-

cular exertion. The individual affected may have enjoyed perfect health up to the date of such exertion; but from that moment has suffered from symptoms of heart affection. We may reasonably conjecture that, in such cases, the tissues of the valves must have been partially degenerated, and, though equal to ordinary pressure, were unable to bear that which resulted from the extra exertion. It is hardly possible to conceive that any degree of inordinate action of the circulating organs could occasion rupture of perfectly healthy valves. The two following cases—and I have notes of several others—will exemplify the point here spoken of:—

A. B., a gardener, about 54 years old, had always enjoyed excellent health until about twelve weeks ago. He dates his illness from a "strain," which happened to him when wheeling a heavy wheel-barrow. From that moment he has never "been himself," and his breathing has been affected. He was, at the time he came to me, suffering from decided symptoms of heart-disease; and auscultation pointed out defect of the aortic valves, there being a loud, diastolic murmur audible over the site of the aortic valves, and down along the sternum. There can be no rational doubt, that in this case the aortic valvular injury occurred at the time of "the strain."

In another case, the subject of the aortic valvular disease was a man about 45. He had always been

very active, and was an excellent runner, and, in fact, used to boast of his wind. About eight months before his death, while running quickly on a message, he was suddenly seized with violent pain in his left side, and in consequence with difficulty reached his home again. From this moment his breathing became affected. I saw him about five months afterwards, and found marked signs of hypertrophy and aortic valvular disease. He eventually sank, after suffering many most severe angina-like attacks. In this case, after death, I found the aortic valves all incompetent; two of them were partially destroyed by ulceration; two of them, also, were puckered, and fused together at their adjoining angles. The aorta, the lining membranes, and the other valves of the heart were perfectly healthy. The heart was greatly hypertrophied. There was no other disease of parts. Is it not fair to conclude, that in this case the heart-disease dated from the time of the great exertion made in running? His breathing had been previously good, and afterwards always bad. There was no other cause to account for the disease; no rheumatism, or kidney, or other affection, &c.

Valvular diseases we therefore find have different modes of origin. They may be the consequences of inflammatory action, and they may be the consequences of textural degenerations. Inflammation softens the valves, in part destroys their tissues, and

so renders them liable to ruptures and bulgings. Its exudation into them gives rise to fibrinous formations, whereby they are thickened, and rendered less pliable, firm, and elastic than natural. Hence arise contractions of the valves, of their tendons, and of their orifices, and adhesions of them to each other, and to the cardiac walls. The fibrinous vegetations (polypoid growths) which form upon their edges, surfaces, and around their tendons, also project between the valves, and prevent their perfect closure. Atheromatous and calcareous degenerations of the valves cause them to assume most varied forms. Sometimes mere specks of the abnormal deposit are seen scattered in the tissues, and sometimes the valves and the arteries are through it converted into semi-solid structures. The calcareous matter, again, may perforate the valves, and assume the most fantastic shapes; thereby, and by deposit of it in the surrounding tissues, the aortic orifice, for instance, may be rendered scarcely large enough to admit the passage of a quill; and so likewise, and by adhesions, the mitral valves may be reduced to the condition of a solid cone, projecting into the left ventricle, having an opening through its apex scarcely large enough to admit the point of the little finger. The destructive tendency of these degenerations, and the degree of obstruction to the circulation, and of regurgitation of the blood which they occasion, our pathological museums abundantly illustrate.

Defects of the valves, I may add, may also result from original malformations of them. Thus, for instance, there may be one valve only at the aortic orifice, or two of the valves may adhere together, or the three may be present, and one of them very small and in a rudimentary state. Congenital defects of the valves of the pulmonary artery are generally associated with other serious organic defects of the heart or its vessels, such as are incompatible with prolonged life. They are therefore rarely met with, except in the very young.

vessels, independently of any endocardial inflammation, in all those diseases in which there is an increase of the fibrin in the blood. This tendency, therefore, especially exists in cases of rheumatic carditis. There are also other diseased states of the body in which this formation of coagula is apt to occur, as when the motion of the blood is impeded, or after loss of blood, or syncope, or when the blood through wasting disease is in an impoverished condition, &c. Attention has of late been particularly called to this subject by the works of Virchow (*Gesammelte Abhandlungen*, p. 219) and Dr. Richardson (*On the Blood*); and to these authors I would refer those who wish to investigate it more fully.

Fibrinous deposits occur in the arteries, the veins, and the heart, but their most ordinary seat is in the heart. That they are frequently formed during life is certain, and that they are occasionally the immediate cause of death is also certain.* Sometimes the deposit gradually accumulates in one part, and so at length arrests the circulation there; sometimes, but more rarely, the fibrinous mass thus formed is carried along in the current of blood, and being arrested in

* "The proof of a *post-mortem* concretion is its position on the upper surface of a red coagulum. The proofs of an *ante-mortem* clot are—the fact of its filling a cavity—the fact of its being grooved externally by a blood current, or bored by a current through its centre—its being firmly adherent to the heart, either by mechanical or organic tie—its structure being laminated, or containing in its centre broken-up fibrin—the fact of its being deeply indented by surrounding structures."—*Dr. Richardson.*

its course blocks up the flow of blood to some organ, the brain it may be, or the lungs. The right auricle is the most favourite seat of these formations in the heart. They always have mechanical attachments more or less firm to the walls of the organ; and are of more or less firm consistence, according to their age. From the deposit, prolongations often pass onwards in the stream of the blood, preventing the proper action of the valves, and blocking up more or less completely the arteries into which they reach. Dr. Richardson first pointed out the fact, that these fibrinous deposits sometimes assume a cylindrical form in the arteries and infundibular portion of the heart and ventricles.*

It must not, however, be forgotten, that fibrinous deposits may form in the arteries, where they are found; for the same causes which excite a deposit of the fibrin in the larger branches of arteries, or in the heart, may equally operate in the smaller branches. It is not necessary, therefore, to suppose that in all cases where vessels of the brain or of the lungs, for instance, are found to be filled with fibrin,

* Dr. Simpson, in his "Memoirs," has given some interesting details in relation to this subject. He tells us (vol. ii. p. 35) that—

"Arterial obstructions may arise from: 1, The separating of old organized cardiac vegetations; 2, The passing into the current of recent fibrinous masses formed in the heart or large vessels; 3, Local arteritis; 4, Laceration of the inner coats of the vessel occluded; 5, Morbid materials carried from the systemic veins into the pulmonary artery."

vessels, independently of any endocardial inflammation, in all those diseases in which there is an increase of the fibrin in the blood. This tendency, therefore, especially exists in cases of rheumatic carditis. There are also other diseased states of the body in which this formation of coagula is apt to occur, as when the motion of the blood is impeded, or after loss of blood, or syncope, or when the blood through wasting disease is in an impoverished condition, &c. Attention has of late been particularly called to this subject by the works of Virchow (*Gesammelte Abhandlungen*, p. 219) and Dr. Richardson (*On the Blood*); and to these authors I would refer those who wish to investigate it more fully.

Fibrinous deposits occur in the arteries, the veins, and the heart, but their most ordinary seat is in the heart. That they are frequently formed during life is certain, and that they are occasionally the immediate cause of death is also certain.* Sometimes the deposit gradually accumulates in one part, and so at length arrests the circulation there; sometimes, but more rarely, the fibrinous mass thus formed is carried along in the current of blood, and being arrested in

* "The proof of a *post-mortem* concretion is its position on the upper surface of a red coagulum. The proofs of an *ante-mortem* clot are—the fact of its filling a cavity—the fact of its being grooved externally by a blood current, or bored by a current through its centre—its being firmly adherent to the heart, either by mechanical or organic tie—its structure being laminated, or containing in its centre broken-up fibrin—the fact of its being deeply indented by surrounding structures."—*Dr. Richardson.*

its course blocks up the flow of blood to some organ, the brain it may be, or the lungs. The right auricle is the most favourite seat of these formations in the heart. They always have mechanical attachments more or less firm to the walls of the organ; and are of more or less firm consistence, according to their age. From the deposit, prolongations often pass onwards in the stream of the blood, preventing the proper action of the valves, and blocking up more or less completely the arteries into which they reach. Dr. Richardson first pointed out the fact, that these fibrinous deposits sometimes assume a cylindrical form in the arteries and infundibular portion of the heart and ventricles.*

It must not, however, be forgotten, that fibrinous deposits may form in the arteries, where they are found; for the same causes which excite a deposit of the fibrin in the larger branches of arteries, or in the heart, may equally operate in the smaller branches. It is not necessary, therefore, to suppose that in all cases where vessels of the brain or of the lungs, for instance, are found to be filled with fibrin,

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that some portion of fibrin has been carried from the heart or elsewhere, and being at length arrested in its course, in a vessel too small to give it passage, has there acted like a plug, and so caused the deposit of fibrin or coagulation of blood behind it. There can be no doubt, that such sudden local arrestments of the circulation do occasionally result from small portions of fibrin—emboli—thus carried along from one part of the vascular system to another; but obstructions from this cause are, I believe, comparatively rare.

The *symptoms* depending upon the formation of clots in the heart differ according to the situation of the clots. When they obstruct the circulation through the right side of the heart, the return of blood from the systemic veins is prevented; the blood does not pass to the lungs for aëration, and, consequently, arterial blood is not supplied to the different organs. Death consequently results, in such case, rather from syncope than from asphyxia. There is also something very remarkable in the extremely painful "cardiac" kind of dyspnœa which attends this affection; it differs from all ordinary kinds of impeded respiration. The formation of the clot in the left side of the heart is indicated chiefly by signs of extreme congestion of the lungs—of ordinary congestion of the lungs, expectoration of mucus and bloody frothy mucus. In this case, if the patient dies, it is rather from coma than from syncope. Of

course, also, there are cases in which clots are formed during life in both sides of the heart.

Of the physical signs attendant upon the formation of clots in the heart, there are none that can be considered as specially pathognomonic. The sudden appearance, however, of a murmur, in cases where no signs of endocarditis existed, and where the symptoms are indicative of formation of a clot, may be considered as an almost certain sign of the fact.

I have not entered particularly into the details of this affection, because the state of our knowledge upon the subject is not yet sufficiently advanced to enable me to speak dogmatically upon it.

I have, however, given in Appendix (No. II.) striking illustrations of the two kinds of affections here spoken of as arising from the formation of coagula—one, in which the coagulum was formed in the heart and extended into the pulmonary vessels; and the other, in which the coagula escaped from an aneurismal sac, and partially blocked up the cervical and cerebral arteries.

CHAPTER VIII.

THE CONSEQUENCES AND GENERAL SYMPTOMS OF VALVULAR DISEASES.

REGURGITATION.—OBSTRUCTION.—PULMONARY, ABDOMINAL, AND CEREBRAL SYMPTOMS.

THE pathological changes of the valves of the heart, above described, occasion impediments to the circulation of the blood through the heart in two different ways. They prevent the valves duly coming together so as properly to close their respective orifices; in consequence of which the blood regurgitates through them. When the mitral valves, for example, are defective, the blood regurgitates into the left auricle during the ventricular systole; and when the aortic valves are defective, from the aorta into the left ventricle, during the ventricular diastole. Then, again, the onward current of blood is obstructed by the narrowing of the orifices of the heart, and by the contractions, &c., of its valves.

Defects of the valves are occasioned in many various ways. They result from their softening, ulceration, rupture, and perforation; the deposit

within them, of fibrinous, atheromatous, and calcareous matters, whereby contraction, hardening, thickening, and rolling up of their edges, is occasioned; and through the deposit, upon them, of fibrinous vegetations. The action of the valves, also, is rendered imperfect by contractions, rupture, adhesions, and thickenings of their tendinous chords, and by all those diseases, also, which interfere with the action of the papillary muscles, and muscles of the heart—by rupture, and fatty and fibrinous degenerations of the papillary muscles, and by extensive dilatation of the ventricles.

The most common causes of *obstructive diseases* are: Rigidity of the valves, and of the tissues around their attached borders, arising from deposits within the endocardium and the valves; Coalescence of the valves to each other and the parts around them; And cretification of the coats of the arteries.

The effect, however, which immediately results from these valvular diseases—viz., impeded circulation of blood through the heart—is much the same, whether it depend upon insufficiency or upon obstructive disease of the valves. The degree of obstruction which the circulation suffers depends upon the amount of change in structure which the valvular apparatus has undergone; and also upon the particular valve which is affected. The injury of the valve—indicated by a permanent cardiac bruit—may be so slight as to betray to the subject of it no

symptoms of its existence; or it may be such as only to indicate its presence when the heart's action is increased by mental or bodily exertion; or, again, it may be so extensive as to render prolonged existence impossible. Then, also, defective states of the auriculo-ventricular valves are of more serious importance—more immediately prejudicial to life—than defective states of the semi-lunar valves. Defective circulation resulting from diseased aortic valves, may be compensated for by healthy hypertrophy of the left ventricle: and under such circumstances, so long at least as the mitral valves remain sound, life may be preserved.* But when the mitral valves are seriously defective, no such compensation is possible, and congestions, &c., of the lungs, which are its most immediate and necessary consequence, readily arise, and so render life precarious.

* The following is a good illustration of this fact:—I have had under my constant observation during the last ten years a man about 34 years of age. During all this time he has suffered from defective aortic valves and hypertrophy of the heart, a loud diastolic murmur being heard down the sternum, and an extensive heaving with the impulse, and dull percussion over the præcordia. The mitral valves are sound. He is, however, at this moment, and has been all along, with the exception of short intervals, able to go about his work (French polishing) as well as any other man. It is worth noticing that, during the time spoken of, this man must have taken about two gallons of the tincture of sesquichloride of iron, and one and a half gallon of tincture of digitalis. He has, again and again, for many weeks together, tried to leave off these medicines; but when he does so, he feels, as he says, that he is fit for nothing.

What are usually known as symptoms of heart disease, are in reality symptoms of the general disorders which result from the obstruction of the circulation here spoken of. These disorders, indeed, often indicate to us, by their severity, the importance of the valvular disease much more than the auscultatory signs do. They are manifested in the heart itself, in the lungs, the brain, the abdominal organs, and generally in the different parts and organs of the body.

Pulmonary Symptoms.—The intimate anatomical and functional relations existing between the lungs and the heart, explain why the effects of an impeded circulation of blood through the left side of the heart should be immediately resented by the lungs. The blood no longer passes freely from these organs, along its wonted channels, into the left auricle of the heart, its passage being obstructed, either by the constricted valvular opening, or by the blood which regurgitates through the defective mitral or defective aortic valves. Thus obstructed in its course, the blood accumulates in the lungs, whose functions are in consequence deranged. Breathlessness is, in fact, often one of the first symptoms which indicate the presence of heart-disease. Congestions, pneumonia, bronchitis, pulmonary apoplexy, and hæmorrhage, œdema, and emphysema of the lungs, are all of them, more or less frequently, the immediate or secondary results of valvular diseases of the heart; the extent of the

particular disorder corresponding with the degree and amount of the valvular lesion. Congestions of the lungs are apt to take place very suddenly in heart diseases; and often also disappear very rapidly, under care and appropriate treatment. The pulmonary hæmorrhage, the abundant bronchial secretion, the pulmonary œdema, and the pleuritic effusions, which so often accompany these diseases, are all, more or less, natural efforts of the lungs to free themselves from the congestions of their blood-vessels.

Abdominal Symptoms.—The abdominal organs, and the brain, likewise suffer from the consequences of the valvular disease; indeed, there is no organ or part of the body which may not, in extreme cases, be brought under its disturbing influence.

Let us see how this happens. The pulmonary circulation being impeded, as above described, the blood no longer flows freely out of the right side of the heart into and through the pulmonary artery; consequently, it accumulates in the right ventricle and pulmonary artery, and thus presents an obstacle to the return of the blood from the venous system, through the venæ cavæ, into the heart. From such accumulations of blood necessarily result congestions of blood, and their consequences, in those organs and parts to which the effects of such an obstacle are immediately communicated. The most important consequences, thence resulting, fall, in the abdomen, upon the liver, kidneys, and intestines. The liver sometimes rapidly

attains, under these circumstances, a very large size; it may even be felt extending low down into the abdomen, several inches beneath the edge of the ribs. The congestion of the kidneys may give rise to temporary albuminuria, which disappears when the congestion, which occasioned it, is removed. The stomach and intestines manifest various signs of disorders resulting from impeded circulation; amongst which may be mentioned, vomiting, hæmatemesis, and discharges of serum and blood from the bowels. By such discharges, indeed, the oppressed circulation sometimes finds relief in these cases.

When the disease of the heart is far advanced, it renders the congested state of these organs a permanent condition, and consequently gives rise to other and secondary affections. The digestion, and therefore also nutrition and absorption, are deranged; and their permanent derangement entails conditions incompatible with due performance of their functions. Then follow dropsies and effusions into the serous cavities; which, again, cause pressure, and so, by their mechanical effect upon internal organs, tend to destroy life. The nutrition being disturbed, the blood is no longer supplied with its proper materials, and defective respiration prevents it from undergoing due aëration. Hence, not only one, but all the vital functions, are thus, directly or indirectly, disordered, each in its derangement tending to complicate the other, and to the increase

of the general disorder, and so to the destruction of life.

The disturbances of the brain which result from valvular diseases do not, generally, show themselves until the valvular disease is much advanced, and the circulation much embarrassed, except under the slight and passing forms of headaches and occasional giddiness.

The brain, in hypertrophy of the left ventricle, is subjected to two injurious influences; namely, the extra impulse of the heart which forces the blood towards the head, and the obstruction to the return of blood from the head. But the *vis a tergo* of the hypertrophied heart, which forces the blood towards the brain, is not so powerful an agent in the production of serous effusions, cerebral congestions, and extravasations of blood, as is an obstruction—of whatever kind—seated in the heart, which impedes the return of the blood from the brain. We may, indeed, conclude generally, that the extra pressure of the blood on the different organs, resulting from the action of an hypertrophied heart, is less than that which results from the venous obstruction accompanying it; and it is evident, that in almost all cases of hypertrophy of the left ventricle, whatever acts as the cause of the hypertrophy, sooner or later acts also indirectly as a cause of obstruction to the return of the blood to the heart.

It would be very difficult to prove (and, reasoning

from analogy, scarcely possible to believe) that the hypertrophied action of the heart ever sufficed of itself to rupture a healthy vessel of the brain, or even to produce coma and apoplectic symptoms.*

The cardiac dropsies which result from valvular diseases are of a mechanical kind. The position of the fluid varies with the position of the body. Dropsy resulting from renal disease, as is well known, usually shows itself in the first instance in the face and upper parts of the body; on the other hand, the dropsy of heart-disease invariably exhibits its presence first in the lower parts of the body.

* At the same time it is well to note, that the force of the impulse of the heart, as communicated to the peripheral parts of the vascular system, is necessarily increased by the abnormal rigidity of the arterial trunks. And we know that rigidity (calcification) of the aorta is very often met with coincidently with a hardened condition of the cerebral vessels. Therefore it follows, that these diseased and weakened vessels are exposed to an inordinate distending force, resulting both from the hypertrophy of the heart and the rigidity of the aorta and great vessels; and hence we can understand how, under such abnormal conditions, their rupture may be occasioned by the violence of the heart's action.

CHAPTER IX.

THE SOUNDS OF THE HEART, THEIR CAUSES, ETC.

THE HEART'S SOUNDS.—FIRST SOUND.—SECOND SOUND.—VARIATIONS IN CHARACTER.—CAUSES.—ARTERIAL SOUNDS.

The Heart's Sounds.—It would be inconsistent with the objects of this treatise, to enter into any minute discussion concerning the causes which produce the normal sounds of the heart; but, at the same time, it is not possible to enter, successfully, on the consideration of its abnormal sounds, without taking a short review of the physiological history of the sounds of the heart—of their nature and their causes.

Two distinctly different sounds, cardiac or arterial, are heard, when the ear is placed over the præcordial region, the one rapidly succeeding the other, without any well-marked interval. These sounds are followed by a distinct period of silence—a pause. The sounds and the pause correspond to one complete systole and diastole of the auricles and ventricles.

The *first sound* is heard, very nearly synchro-

nously, both with the impulse of the heart against the thoracic walls, and with the beat of the arteries in the neck. It is dull and prolonged, and takes up about two-fifths of the whole interval occupied by the sounds and the pause. The systole of the ventricles, and the diastole of the auricles, take place during the period of this sound.

The *second sound* succeeds the first, without any well-marked intervening pause; it is clear, well-defined, sharp, and of short duration. It corresponds, in time, to a part of the ventricular diastole, and to the receding of the heart's apex from the point, where it impinges upon the thoracic walls during the systole; it occupies about one-fifth, or rather more, of the entire interval before mentioned. It is followed by the pause, which takes up about two-fifths, or rather less, of the whole interval; and during this pause, the auricular systole and the completion of the ventricular diastole take place. The second sound is, as a rule, heard loudest at that part of the thoracic walls which is nearest to the situation of the semi-lunar valves; the first sound, at that part where the heart's impulse is felt.

The following table shows the different physiological facts which occur in correspondence with the sounds and the pause:—

First Sound.	Systole of ventricles. Distension of aorta and pulmonary artery. Diastole of auricles. Impulse.

Second Sound. Passive flow of blood from auricles into ventricles; and first stage of diastole of ventricles. Contraction of aorta and pulmonary artery.

Pause. Continued passive flow of blood into ventricles. Towards the end of pause rapid contraction of auricles, occasioning forcible and complete distension of ventricles, which is instantly followed by a succeeding systole.

The sounds of the heart vary even in healthy individuals, in respect of their strength and clearness, in the extent to which they are heard over the thorax, and also with regard to the points of the thorax at which they are heard loudest. In one person, we find them loud and ringing, in another, scarcely perceptible; at one time, they are heard over every part of the thorax, and at another, scarcely reach beyond the cardiac region. They may be loud at the heart's apex, and weak at its base, and *vice versâ*. They vary, also, according to the age and sex of the individual, being clearer in women and children than in men; and they are affected by mental emotion. They are modified by the position of neighbouring organs; by the thickness of the soft parts which cover the thorax; by displacements of the heart; by encroachment of the lungs over the organ, as in emphysema; by pericar-

dial effusions; by a distended stomach; by tubercular diseases of the lungs, &c.

It must be remembered, that when we are speaking of sounds of the heart, we are referring to sounds which, whatever their cause, have a double origin: there are two ventricles, two sets of valves, two great arterial trunks,—which act alike and simultaneously. Hence, whatever sound is produced, either by them or by the currents of blood passing through the heart, has a double, though a similar origin.

The sounds, also, heard over the præcordial region, and, as they are ordinarily called, cardiac sounds, arise not only in the ventricles, but also in the aorta and pulmonary artery. This is a very important fact, which was first clearly laid down and illustrated by Skoda. He tells us, that "the ventricles, the aorta, and pulmonary artery, severally contribute to the production, both of the first and of the second sound of the heart." As proof of this statement, he calls attention to the following facts: the first sound, as heard over the base of the heart,—that is, near the roots of the aorta and pulmonary artery,—is frequently less marked than the second sound; whilst, on the other hand, at the heart's apex, the first sound is the most accentuated and prolonged. The sounds heard over the apex, that is, over the left ventricle, often differ materially in strength, pitch, and clearness, from those heard over the lower sternum, that is, over the right ventricle.

The sounds heard about the middle of the sternum, and at its right border, that is, over the aorta, often differ markedly from those heard to the left of the sternum, and on the same level, that is, over the pulmonary artery; and this is particularly the case in certain diseased conditions of the heart, when the passage of the blood through the pulmonary artery is obstructed.

Again, cases occur in which the sounds of the heart are plainly audible over the right ventricle, over the aorta and pulmonary artery at the base of the heart, whilst at the apex they are altogether replaced by a double bruit. A bruit, also, may be heard over the right ventricle, and yet both sounds be clear and good over the apex and base of the heart. And it very frequently happens, that both sounds may be audible over the right and left ventricles and the pulmonary artery; and a double or single bruit, at the same time, be heard along the aorta.

The first sound heard over the aorta must be attributed to those efficient causes of sound which are in action nearest to the point where it is heard; and hence, the sudden distension of the coats of the aorta may be its chief source, and the impulse of the heart have little to do with its production. And again, conversely, at the apex of the heart the impulse may account for much, and the distension of the aortic coats for little, in the sound's production.

These facts may be readily applied in other instances; and their consideration is of great value in the matter of diagnosis.

Causes of the Heart's First Sound.—It will assist us much, in considering the sources whence the heart's sounds arise, to keep in view the chief physiological facts which attend, and are coincident in time with, the periods at which the two sounds occur. Premising, that I accept, as an indisputable fact, the proposition, that the first sound coincides with the impulse of the heart; and that, when I speak of sounds of the heart, I include the two sounds, wherever audible over the præcordial region, and consequently those which proceed from the aorta and pulmonary artery, as well as from the heart itself.

The following are the physiological phenomena which are associated with the heart's action, during the period of the first sound:—

1st. The coming together, and the forcible tension, of the mitral and tricuspid valves.

2nd. The muscular contraction of the ventricles.

3rd. The impinging of the heart against the walls of the thorax.

4th. The rush of blood, forced by the ventricular contractions, through the orifices of the aorta and the pulmonary artery.

5th. The falling back of both sets of semi-lunar valves toward the walls of the aorta and pulmonary artery.

6th. The sudden distension of the aorta and pulmonary artery.

It is probable, that each one of these facts, with the exception of the fifth, aids in the production of the first sound of the heart. Science, however, has not yet explained to us the particular share which each takes in its production. At the same time, it must be admitted, for sufficient reasons, that there is one cause, viz.—tension and closure of the auriculo-ventricular valves, which is far above all others effective in producing it.

This conclusion is in accordance with the main characteristics of the sound, with the results of experiments, and is strongly corroborated by this capital fact, derived from clinical observation, namely, that alterations of sound are almost invariably associated with alterations of the structure of the valves. (See Appendix III.)

The Second Sound of the Heart.—The chief acts which occur during the period of the second sound are the following:—

1. The valves of the aorta and pulmonary artery are suddenly closed, and rendered tense.

2. The distended coats of the pulmonary artery and the aorta contract on their contents.

3. The auriculo-ventricular valves separate, and fall back towards the walls of the ventricles.

4. The first part of the ventricular diastole takes

place, and the blood passes into the ventricles from the auricles.

But though these several acts coincide with the period of the second sound, it is now universally admitted, that the chief, if not the sole cause of it, is the tension of the semi-lunar valves of the aorta and pulmonary artery. This opinion is confirmed both by experiment and clinical observation. Experiment shows, that when the semi-lunar valves are mechanically separated during the ventricular diastole, the sound ceases, and is replaced by a bruit. And the same thing is constantly matter of clinical observation, in cases where the aortic valves are rendered incompetent by disease. It is also corroborated by the fact, that the second sound of the pulmonary artery often becomes very loud, when the passage of the blood through its branches is impeded; as happens in severe cases of mitral valvular disease.

Varieties in the Characters of the Heart's Healthy Sounds.—I have already observed, that differences occur in the characters of the sounds of the heart, as heard even in healthy individuals. Of these, however, it is not possible to give any distinct account; they are very numerous, and the conditions which they indicate are, for the most part, obscure.

When the first sound, heard over the ventricles, has a clear, distinct, flapping character, we may generally predicate a healthy condition of the auri-

culo-ventricular valves, their large size, and fineness of structure. This clear character of the sound is lost when the valves are contracted, or their borders thickened.

The reduplication of the first sound, which is occasionally heard over the ventricles, is difficult of explanation; it has been attributed to a want of synchroneity in the contraction of the two ventricles, and to irregularities in the time of closure of the auriculo-ventricular valves. The second sound, heard over the ventricles and over the pulmonary artery and aorta, is also occasionally broken and interrupted; this condition of it has been ascribed to irregular closure of the semi-lunar valves. Moreover, the two sounds are occasionally found to vary much in their duration, and so also are the intervals of silence, which separate them. And I may also add, as worthy of note, that the presence of normal sounds, as well as the absence of bruits, are requisite to indicate a sane condition of the different parts of the heart. Absence both of the normal sounds and of distinctly marked bruits, when other signs and symptoms of heart disease exist, generally indicates a very advanced stage of structural disease.

Arterial Sounds.—Two sounds, synchronous with and resembling the cardiac sounds, are heard over the great arteries, in the neighbourhood of the heart.

The *second* of these sounds is only heard in the vessels near the heart. It corresponds in time with

the ventricular diastole and the arterial systole; and has doubtless the same origin as the second sound of the heart, being produced by closure of the semilunar valves. Pathology corroborates this, for we find, that when the aortic valves are defective the second sound normally heard in the cervical arteries disappears.

The first arterial sound corresponds with the cardiac systole and the arterial diastole. It diminishes in intensity with the size of the arteries; and is modified in timbre, according to the degree of distension of the vessels, their rigidity, thickness, &c. The more powerful the circulation, the further is it heard along the arteries. In the arteries near the heart it is doubtless augmented by the first cardiac sound. It is in all cases intensified, and in the smaller arteries probably produced, by the pressure of the stethoscope upon the walls of the artery, whereby a local contraction of the vessel is occasioned. But it is not difficult to conceive that the sudden distension of vessels of a certain size, and the friction of the blood against their walls, may excite audible vibrations. Sometimes, in cases of anæmia, the slightest pressure upon the radial artery will excite a loud, blowing murmur.

CHAPTER X.

IMPULSE OF THE HEART.

CAUSES.—SITUATION OF IMPULSE.—VARIETIES.

Impulse of the Heart.—During the ventricular systole, the heart's apex is, in health, felt impinging against the thoracic walls in the fifth intercostal space on the left side, at a point about midway between the left border of the sternum, and a line drawn perpendicularly downwards through the left nipple.

The *situation* of the impulse varies slightly, even in health. The heart, being loosely attached in the thorax, follows to a certain extent the laws of gravity; and its impulse is therefore felt more or less to the right or left of a particular point, according to the position of the individual.

Certain diseases of the heart, and abnormal conditions, also, of the parts around it, alter considerably both the force of its impulse, and its point of contact with the thoracic walls. Thus, for instance, hypertrophy and dilatation, and atrophy of the heart, pericardial effusions, pleuritic exudations, pneumo-

thorax, thoracic aneurisms, emphysema of the lungs, mediastinal tumours, enlargement of the liver, and particularly of its left lobe, peritoneal effusions, a tympanitic condition of the stomach and intestines, and hypertrophy of the spleen—all modify, in a greater or less degree, the force and position of the impulse.

A knowledge of the position of the heart's apex aids us in ascertaining the condition both of the heart itself, and of the different parts around it.

The *force* of the natural impulse varies much in different individuals. In large, broad-chested, muscular persons, it is not so readily felt as in those in whom the soft parts of the chest are less developed; sometimes, indeed, it is scarcely perceptible, especially in the recumbent position. During inspiration, also, its force is lessened. The impulse is generally well marked in women and children; and is, in all cases, felt best when the body is inclined forwards, and during expiration,—that is, when the heart, through the contraction of the lungs, is permitted to come more immediately, and completely, in contact with the walls of the chest.

The force of the impulse is increased by muscular exertion, and by mental emotions, in febrile disorders, and in diseased states of the heart itself. Its natural force, again, may be diminished, as in chronic wasting diseases, in emphysema of the lungs, œdema of the thorax, pericardial effusions, &c.

A strong and violent beat of the heart does not necessarily indicate its hypertrophy; it frequently happens, indeed, on the contrary, that a hard, sharp and quick impulse, concentrated at one spot, feeling sometimes like the blow of a hammer, indicates a weakness of the organ. Such a beat is met with often in weak and excitable females, and is apt to mislead the inexperienced observer as to the condition of the heart, which produces it. The impulse of an hypertrophied heart (hypertrophy being usually combined with dilatation) is not concentrated, and has more the character of a diffused heaving, than of a sudden blow; it raises the thoracic walls and the head of the auscultator—the thoracic walls sinking back again, during the diastole of the heart. When the impulse is perceptible in several intercostal spaces,—and it never is so, when healthy, in more than two, —or for some considerable distance along one intercostal space, we may be certain that the heart is enlarged.

An hypertrophied heart, however, may, when quiescent, yield a perfectly normal impulse; and its impulse may also be modified by the effects of pericardial effusion, and other diseases. Consequently, no positive conclusions as to the condition of the heart can be drawn from an impulse which is weak or barely perceptible.

Cause of the Heart's Impulse.—The cause of the heart's impulse has not yet been satisfactorily explained.

The very different theories which have been offered as explanatory of this phenomenon, suggest the idea, that here, as in the case of the heart's first sound, more than one cause may operate in its production.

A careful consideration indeed of the different facts which are associated with the impulse of the heart almost naturally leads us to the conclusion, that it is not a simple act, but that it depends: partly upon the modifications in form and consistency of the heart's walls, which occur during its ventricular systole; partly upon changes produced in the position and form of the heart, as consequences of the contraction; and partly also upon a certain degree of displacement, which the organ itself undergoes, as a result of the ejection of fluid from its interior during the ventricular systole.

The most trustworthy experiments on this subject show, that the heart, during systole, moves from above downwards, and from left to right, and therefore somewhat in a spiral direction. The consequence of this is, that the apex of the left ventricle is brought into contact with the thoracic walls. Kiwisch, Bamberger, and others, argue that it is the hardening and arching forward of this portion of the left ventricle which produce the impulse. This hardening and arching, it is said, may be felt by the hand at all parts of the left ventricle when the heart is grasped by it, but are perceptible only at one point of the præcordial region in the normal condi-

tion of parts, because only the apex of the left ventricle comes in contact with the thoracic walls. The portion of the right ventricle which impinges against the thoracic walls produces, normally, no impulse, because its muscular contractions are less powerful than those of the left ventricle, and also because it lies mostly behind the sternum.

We may also take into consideration here, as confirmatory of this view, that peculiar arrangement of some of the strong bands of muscular fibres of the heart, which was pointed out by Dr. John Reid in his well-known article on the heart in the *Cyclopædia of Anatomy*. From the mode of attachment of these fibres, the apex of the heart is necessarily drawn forwards during systole. That the projection of the blood from the ventricles may also assist in producing or modifying this impulsive movement of the heart seems far from improbable.

From the above considerations, thus shortly alluded to, I conclude, that what is called the impulse of the heart is the result of several combined facts. (See Appendix IV.)

CHAPTER XI.

ENDOCARDIAL MURMURS.

MURMURS.—CAUSES OF ENDOCARDIAL MURMURS.—THE PARTS WHERE THEY ARISE.—THEIR VALUE IN DIAGNOSIS.

Murmurs, Bruits—abnormal sounds—heard over the præcordial region, synchronously with the movements of the heart, and replacing or co-existing with its normal sounds, indicate, with few exceptions, that certain portions of the heart, or of its great blood-vessels, have undergone organic changes. These bruits are most important diagnostic signs of the chief forms of heart diseases. They are the consequences, and therefore the indicators, of abnormal conditions of parts within the heart, of diseased states of its valves, of the valvular orifices, and of the great arteries, in the immediate neighbourhood of the heart.

I have already given an account of the abnormal sounds which are produced by pericardial disease; the attention of the reader will, therefore, in this place be confined to a consideration of the bruits which arise within the heart, the aorta, and the pulmonary artery—that is, to endocardial and arterial bruits.

Endocardial bruits present themselves to us under a great variety of characters, and are described as blowing, sawing, whistling, purring, rasping, bellows-like sounds. There is no practical advantage, however, to be derived from an attempt to define, too nicely, all their particular resemblances. The character of the murmur, in any given case, is not a fact of much importance to arrive at; and, indeed, it is in most cases rather arbitrarily defined, depending somewhat upon the ear of the observer, and upon the particular state of the heart at the time of the stethoscopic examination.

Different observers will be often found to give different descriptions of the same murmur; and the same endocardial lesion, indeed, may, under opposite conditions of the heart's action, produce very different kinds of bruits. Thus, a murmur which is of a gentle blowing character when the heart's action is quiet, may quickly pass into a rough sawing murmur on any sudden increase of its action. Or, again, a loud murmur may be heard over the heart when acting violently, and disappear altogether during its tranquil movements. And in some of the worst forms of endocardial disease, in their latter stages, when the heart's action is enfeebled, and its cavities distended, the bruits sometimes entirely disappear.

Hence, then, we may conclude that the nature and extent of the endocardial injury cannot be learnt from any character of the bruits thence arising. All

that can be fairly said of them is, that the rougher and more loudly vibratile the bruits are, the more well marked, as a rule, are the organic changes which have occasioned them. The all-important point in diagnosis is, first, to ascertain with certainty the existence of the bruit, and then, to discover the point within the heart, or great vessels, where it arises.

Causes of Endocardial Murmurs. — Endocardial bruits, which either entirely supersede, or partially replace, the normal sounds, are almost invariably found to be associated with a diseased or defective condition of the valves, or of the valvular orifices of the heart. When these parts are disorganized, the current of blood no longer runs its smooth, uninterrupted course through the heart; the stream is obstructed, or its direction is altered. The unnatural friction of the blood against the valves, or the walls of the ventricles, or of the great arterial trunks, excites unnatural vibrations, which occasion the murmurs.

The pathological changes which give rise to these unnatural states of the current of blood within the heart and the great vessels, and consequently to its murmurs, have been already described.

I may here note as worthy of remembrance, that very exceptional cases are occasionally met with, in which loud bruits have been heard during life, and in which, nevertheless, either no structural changes

have been found after death, or not such as seem sufficient to account for the bruits. In considering the meaning of all such anomalies, we must remember, that we have no means of deciding accurately as to the perfect closure or otherwise of the auriculo-ventricular valves after death, for we cannot perfectly imitate the muscular movements of the heart.

Whether, again, an endocardial bruit can result from an open foramen ovale, has not yet been determined. A case described by me in the *Pathological Transactions* (vol. viii. p. 142) seems to indicate that, under certain conditions of the opening, such a bruit is possible. In the great majority of cases, however, it is certain that the condition of the opening is of a kind which is not capable of exciting a murmur.

Seat of Endocardial Murmurs.—Particular rules have been laid down for our guidance in the diagnosis of valvular diseases, to enable us to ascertain the exact seat of each murmur; and in the majority of cases we may safely trust to their guidance.

It is only right, however, to warn the student, that these rules are not universally applicable, and that he will meet with anomalies in this part of the history of cardiac murmurs. This he may well expect to do, if he recollect what has been said respecting the obscurity which still envelopes the subject of the causes of the sounds of the heart. Happily, a very exact knowledge of the seat of a murmur is not essential for our guidance in the practical application

of our art to the treatment of cardiac diseases; and upon this fact I would lay especial stress at the present moment, when so much minute consideration is given to the differential diagnosis of valvular diseases of the heart.

It is of infinitely more importance for us to ascertain the fact, that a murmur, indicative of organic lesion of some of its parts, exists in the heart, than to know *where* the murmur takes its rise. The fact of the existence of the lesion being determined, our treatment of the whole train of general symptoms which exist, either in coincidence with, or as the consequences of, the cardiac lesion, is in no way assisted by a knowledge of the exact situation of the disease which produced the symptoms. In cases, for instance, of chronic valvular diseases, we are not called upon to treat the valvular disease itself, but to deal with the symptoms which are its natural consequences; and in acute diseases of the valves,—associated with rheumatic fever, for example,—the actual situation of the endocardial disease in no way whatever regulates our treatment of it.

Obstructive diseases of the valvular apparatus,—constrictions of their orifices, however produced,—as a rule, give rise to more severe general symptoms, or in other words, to greater impediments to the circulation of blood through the heart, than diseases in which the valves are simply defective; that is, in which regurgitation takes place. But in some cases

the valvular defect may be so great as to render the regurgitation as much a source of impediment as obstructive disease usually is. Hence, in such cases, where the grand fact—the impeded circulation—is manifest, the cause thereof becomes of very secondary importance as regards their treatment. Whether the murmur be mitral or aortic, whether it be diastolic or systolic, can matter but little to the practitioner; its nature in such case neither influences nor directs his treatment.

Not that I would desire to undervalue the advantages of a careful differential diagnosis of endocardial diseases; for the very habit of scrupulously investigating their physical characters, leads the student, necessarily, to a more intimate acquaintance with their nature. What I desire to inculcate is this, that a too curious investigation of physical signs is apt here, as in the case of all other diseases wherein physical diagnosis is concerned, to make the observer less attentive to the general signs and symptoms than their real importance demands, and thus to induce him to give more weight to the physical signs than they reasonably merit. The local physical sign is most valuable as an indicator of the heart disease; but in the treatment of the disease it is, perhaps, the least important of the coexistent signs and symptoms. Thus, a murmur may demonstrate the presence of disease in the heart, and no other signs or symptoms exist to corroborate the fact: in

such case the value of the murmur is great, because it tells us that the heart is affected, and consequently enables us to anticipate and guard against future evils; but it points out no special treatment, and the injury which its existence indicates (supposing all other signs and symptoms of cardiac disease absent), demands none. Again, the same murmur may be present, and, with it, a train of symptoms which indicate great obstruction to the circulation; in this case also, the value of the murmur is great, because it points out to us the original source of the disorders, but it indicates no special treatment.

CHAPTER XII.

INORGANIC MURMURS, ETC.

CARDIAC AND ARTERIAL.—THEIR CAUSES.—THE PARTS WHERE THEY ARISE. — MURMURS OVER THE PULMONARY AND SUBCLAVIAN ARTERIES.—VENOUS MURMURS.

Inorganic Murmurs.—There are two sources of error, which may mislead us, in judging of cardiac murmurs as indicators of structural diseases of the heart. In the first place, murmurs unassociated with any distinct organic changes of parts, are occasionally heard during life over the heart and arteries. And secondly, serious organic changes may exist, and yet give rise to no cardiac murmurs. From hence it follows, that neither is the absence of murmur a certain indication of absence of valvular disease, nor is the presence of murmur, *per se*, a sure sign of the existence of organic disease.

It appears probable that inorganic murmurs, heard over the heart synchronously with the ventricular systole, are, in most cases, not truly cardiac, but arterial or venous murmurs; in fact, that they arise either in the large venous trunks, in the aorta, the pulmonary artery, or at their arterial or venous orifices, and not within the heart itself. In the great majority of cases it is certain that inorganic mur-

murs have the characters of systolic aortic murmurs; and only exceptional instances give colour to the belief that mitral systolic murmurs are of inorganic origin. We may take it as a rule, having few if any exceptions, that mitral systolic murmurs indicate a defective condition of the mitral valves.

Inorganic murmurs, heard at the præcordial region, have the characters, in general, of arterial murmurs, —they are soft and blowing. They usually exist in cases in which murmurs may, at the same time, be readily excited in the larger arteries of the body, and in which the blood has undergone changes, either qualitative or quantitative; or in cases where the proper relations, which should exist in health between the vessels and their contents, are lost. Inorganic murmurs, moreover, are always systolic, and are rarely ever heard elsewhere than at the base of the heart; seldom do they reach below the nipple, and are, perhaps, never audible at the heart's apex. Again, a mitral systolic murmur is necessarily a regurgitant murmur; and a regurgitant murmur, unassociated with some defect of the mitral valves, is a condition, in the present state of our knowledge, scarcely comprehensible.* And, as already observed, mitral—

* That certain spasmodic contractions of the papillary muscles may, at times, occur, and so prevent the closure of these valves for a certain period, I can neither believe nor deny, having no data to guide me to an opinion on the subject; but, assuredly, there is nothing of a spasmodic character in the continuous, uninterrupted smoothness of ordinary inorganic murmurs. The

so-called—inorganic murmurs are admitted, by those who believe in their existence, to be of very rare occurrence, whilst arterial inorganic murmurs are notoriously common.

For these reasons, we may fairly admit the general conclusion,—that inorganic murmurs heard over the heart are of arterial origin; that is, arise either within, or at the orifices of, the aorta and pulmonary artery, or in the large venous trunks.

Inorganic murmurs arise in the course of various diseases,—in chlorosis, in anæmia, and in most diseases which produce an impoverished condition of the blood; they may arise also after great loss of blood.* Occasionally, also, systolic basic murmurs are present, temporarily, in the acute stages of many inflammatory diseases,—in typhus, in the exanthemata, in pneumonia, in acute rheumatism, and other disorders. The name of *hæmic* has been given to these murmurs, and with some show of reason, inasmuch as they appear, for the most part, to arise in connexion with those diseases, in which there exists a deviation from the healthy constitution of the blood.

murmurs which are occasionally heard in cases of chorea, and which apparently disappear after a time, are of too obscure a nature to be taken into consideration, as examples of inorganic mitral murmurs.

* It is, I think, worthy of note, that in the anæmia which arises as a consequence of organic disease of the heart, a venous murmur, the *bruit de diable*, is rarely to be met with. I do not know whether other observers have come to a similar conclusion.

The true cause of inorganic murmurs has not yet been clearly determined. They have been attributed to the watery state of the blood; the supposition being, that the particles of the blood, under such circumstances, are readily thrown into vibrations. It has also been supposed that the murmurs may result from the sudden and quickly performed ventricular contractions of the heart, which are so often observed in spanæmia—the violence and rapidity with which the blood is driven through the arterial orifices, producing the same results as ensue from contraction of the orifices under ordinary conditions of the heart's action. This view derives some support from the fact, that anæmic cardiac murmurs, heard when the heart's action is violent, sometimes disappear when its action is tranquillized. Another hypothesis is, that the due relation as regards pressure, which exists in health between the blood and the heart and vessels containing it, is lost—the coats of the vessels having lost their tonicity through defective innervation.

In conclusion, I would observe that murmurs heard in the neighbourhood of the heart are not always cardiac murmurs. Murmurs, for instance, are not unfrequently heard under the left clavicle in tubercular disease of the lungs; and they have been ascribed to pressure of the hardened lung upon the left subclavian artery (Dr. Kirkes)—an explanation which may be well accepted in some cases. I have

frequently seen the first portion of the subclavian artery lying in a deep groove or channel, which it had formed in the consolidated apex of a tubercular lung.

Systolic murmurs are also not unfrequently heard in the second left intercostal space over the situation of the pulmonary artery; but no complete account of their cause has yet been given. It is certain, however, that they sometimes exist in connexion with tubercular disease of the lungs. Sometimes also the least pressure upon the subclavian artery in persons who are slightly anæmic will occasion a very loud murmur; and in strong muscular men a loud bruit is often heard in the subclavicular region, excited in the subclavian artery, and probably by pressure of the muscles upon the artery.

It is possible, also, that a murmur coincident with the systole of the heart may be excited by pressure of a consolidated portion of lung upon a branch of the pulmonary artery, within the lung itself, and also by the pressure of enlarged bronchial glands upon the roots of the artery. In a little girl who was suffering from inflammatory consolidation of the lower half of the left lung, I heard a clear and soft systolic murmur over a space of about two inches diameter below the left scapula. There was no bruit whatever audible over the cardiac region; but both sounds of the heart were distinct and good there, and likewise over the upper half of the scapular region, and over the right

side at the inner border of the right scapula. This murmur was persistent for several days, and was manifestly connected with the consolidation of the lung; for when the child recovered from the pneumonia, the bruit entirely disappeared. I conclude that it must have been occasioned by pressure upon some branch of the pulmonary artery within the lung.

The respiratory murmur itself, when coincident with the systole of the heart, may be, and often is, mistaken by the inexperienced observer for a cardiac bruit, especially when the respiration is hurried and jerking. I have even known the loud and rapid murmurs heard over the upper part of the sternum, and accompanying respiration and expiration in a nervous female, who was breathing about sixty times in a minute, mistaken for a double aortic bruit. Of course, murmurs thus excited will at once disappear when the person auscultated is made to cease breathing for a few seconds.

I may also add, that in cases of anæmia, loud venous murmurs, the *bruit de diable*, are often audible under the sternal end of the clavicle, and over the roots of the large venous trunks, somewhere about the union of the subclavian and internal jugular veins. Such murmurs are readily distinguished from arterial murmurs by being continuous, and by the peculiar character of the sound they produce.

CHAPTER XIII.

ORGANIC ENDOCARDIAL MURMURS.

ORGANIC ENDOCARDIAL MURMURS.—SEAT OF THE MURMURS.—SYSTOLIC MURMURS OVER THE LEFT VENTRICLE OF THE HEART.—REGURGITANT MITRAL MURMUR.—DIASTOLIC MITRAL MURMUR.—SYSTOLIC AORTIC MURMUR.—REGURGITANT AORTIC MURMUR.—DOUBLE AORTIC MURMUR.—TRICUSPID MURMUR.—PULMONARY ARTERY MURMUR.

All abnormal sounds arising through disorganization or visible alteration of the heart's structure, which are heard over the cardiac region, and proceed from, or arise within the heart itself, at its orifices, or within the trunks of the aorta or pulmonary artery, are included under the head of organic endocardial murmurs.

Endocardial murmurs are usually heard loudest at those parts of the thoracic walls which are nearest to the points whence they take their origin; but to this rule there are exceptions. They correspond closely, in time, to the systole and diastole of the heart, but their duration does not always correspond with the natural length of the heart's sounds. The diastolic, for instance, in some cases becomes longer than the systolic sound, and the pause which naturally succeeds the diastolic sound, almost imperceptible. The rhythmic movements of the heart are,

in fact, deranged through the organic valvular disease which occasions the murmurs.

Murmurs are heard most readily in the direction of the current of the blood in which they are produced. A systolic aortic murmur, for example, passes most freely upwards along the aorta; a diastolic aortic murmur most freely downwards, towards the ventricles. But though murmurs pass most freely in particular directions, it is well to note that the vibrations, which excite the sounds, spread in all directions, and therefore also in an opposite direction to the current of blood; so that murmurs, when intense, sometimes pass even to a considerable distance from their seat of origin, in a direction contrary to the current of blood.

Endocardial murmurs, again, may be heard simultaneously with the heart's healthy sounds,* which are then modified in their characters; or they may entirely replace and supersede these sounds; or one of the heart's sounds may commence as a murmur and terminate as a healthy sound. Organic murmurs, also, are persistent, not temporary, like the inorganic sort; but it must be remembered that, in some cases, a murmur is only audible when the heart's action is inordinately excited. In the latter stages of heart diseases, also, when the heart's action

* I may as well observe here, to prevent any chance of confusion, that when I use the term *sounds* of the heart, I refer to its healthy sounds.

is enfeebled, and its cavities distended and oppressed with the blood, murmurs, previously existing, sometimes cease to be heard; but in such case, together with absence of murmur, there will be absence of the healthy sounds of the heart.

Seat of the Murmur.—In order to determine the point of origin of any particular murmur, we must find out the parts of the thorax at which the murmur is heard loudest, and the direction in which it is conveyed. Now, as almost all cardiac murmurs arise at or about the orifices or the valves of the heart, and are, *cæteris paribus*, heard loudest nearest to their points of origin, it is important that we should ascertain, as closely as possible, the position of the valves and the orifices. This it is sometimes difficult to do; for casual circumstances, even in health, tend somewhat to change the position of the heart; and when the shape and figure of the organ is altered by disease, its position, and the relation of its individual parts to the thoracic walls, become still more deranged.

We shall find, however, in the point where the apex of the heart beats against the thoracic walls, an important guide for determining the relative position of its other parts. This point can almost always be ascertained; and it represents the situation of the apex of the left ventricle. Consequently, as a general rule, a cardiac murmur, heard loudest at that point (when the apex is felt in its natural posi-

tion), must be ascribed to the left side of the heart; and in like manner, a murmur heard loudest over the lower part of the sternum, must be attributed to the right side of the heart. Murmurs heard loudest about the centre of the sternum, and passing upwards a little to the right of it, belong to the aorta, as a rule; and those heard to the left of the sternum, about the second intercostal space, to the pulmonary artery.

Systolic Murmurs over the Left Ventricle of the Heart.—A murmur, heard about the apex of the heart, and in a direction upwards, towards the left of the nipple, indicates,—either imperfect closure of the mitral valves, and, consequently, regurgitation of blood into the left auricle; or, the existence of irregularities or roughnesses—from calcareous or fibrinous deposits—upon the surface of the endocardium near the valvular orifices, or upon the ventricular surfaces of the aortic valves, causing obstructions to the free current of blood from the ventricle into the aorta.

When the systolic murmur results from *imperfection of the mitral valves,—systolic or regurgitant mitral murmur,*—it is heard loud at the apex of the heart, and in the direction of the mitral valves; that is, upwards, and to the left of the nipple. The murmur may be heard very faintly, or not at all, over the situation of the aortic valves, and up the sternum, and is sometimes strictly limited to the parts about the left of the left nipple. It is gene-

rally, however, when very intense, and when the heart is much enlarged, audible over a large portion of the præcordial region. Both the cardiac sounds over the aorta may, at the same time, be heard clear and healthy; and so, likewise, both sounds over the lower part of the sternum,—that is, over the right ventricle and right base of the heart. The murmur, however, may be heard in these situations, but then it will be much less loud than over the left ventricle. Regurgitant mitral murmur, also, sometimes entirely supersedes, or partially replaces, the first sound at the apex; and is frequently audible, more or less distinctly, in the lower part of the left interscapular space.

When the murmur results from the friction of the blood against the roughened surfaces of the endocardium, at and around the arterial orifice and its valves, or from constriction of the arterial orifice, it is heard indistinctly, and sometimes not at all, at the apex of the heart, but becomes more distinctly audible as we proceed upwards from the apex towards the mid-sternum,—that is, towards the site of the aortic valves, which is its point of intensity, and from whence it is conveyed upwards, in the direction of the aorta, either partially or wholly replacing the first aortic sound. Consequently, the point of intensity of a murmur, thus arising in the left ventricle, lies quite in a different direction to that of a mitral regurgitant murmur.

The condition of the second sound of the pul-

monary artery has been proposed as a test by which to decide, as to whether the ventricular murmur depends merely upon friction of the blood against the roughened surfaces of the ventricle, or of the ventricular surfaces of the valves; or whether it is the actual result of mitral regurgitation. Skoda, indeed, lays it down as a rule, that we are not to conclude, from the mere fact of a murmur being heard in the left ventricle during the heart's systole, that the mitral valves are defective, unless we at the same time ascertain that there is an increase in the intensity of the pulmonary artery. The increase of its sound is accounted for in the following manner:—"When the closure of the mitral valves is not complete, a portion of blood, at each systole, regurgitates from the left ventricle into the left auricle, causing distension of the left ventricle and of the pulmonary veins and arteries, so that increased efforts on the part of the right ventricle become necessary, in order to force the blood onwards through the distended vessels; the pulmonary artery, thus strongly dilated, presses with the increased force of its elasticity upon the blood within it, and drives it more suddenly and forcibly backwards than ordinary, against the semilunar valves, during the heart's diastole, whereby the second sound of the pulmonary artery is intensified." (Skoda.)

I believe the value, as a diagnostic sign, of this increase of the second sound of the pulmonary artery,

has been much overrated by the Germans; but at the same time I admit that, in most cases, where there is considerable mitral regurgitation, the sound is really intensified. In these cases, however, marked oppression of the lungs also coexists, and tends to point out the nature of the murmur, equally as the increased second sound; and therefore reduces its value as a diagnostic sign.*

The right ventricle and the left auricle are usually more or less hypertrophied and dilated when the mitral valves are defective; consequently, the extent of præcordial dulness is found on percussion to be greater than natural. The character of the pulse varies considerably in such a condition of the heart; when the regurgitation is considerable, it becomes weak, irregular, and unequal, on account of the small and varying quantity of blood which is thrown into the aorta during each ventricular systole.

* The sounds of the pulmonary artery are to be sought for in the second left, those of the aorta in the second right intercostal space, about a finger's breadth from the sternum. In health, the strength, pitch, timbre, and duration of these two sets of sounds are generally alike, but occasionally they vary. Whatever produces increased tension of the pulmonary artery, produces increase of its second sound. So Hamernjk writes of the second sound of the pulmonary artery. I have paid some attention to this second arterial sound, and may say of it, that I have never been able to decide whether, in health, the second sound of the aorta, or of the pulmonary artery, is the louder. I have found the intensity of the two sounds vary much in different persons, being sometimes greatest in the aorta, and sometimes in the pulmonary artery.

The cause of hypertrophy and dilatation of the right ventricle and left auricle in such cases is evident enough. But the cause of the hypertrophy of the left ventricle, as a consequence of defect of the mitral valves, is not so clear. It has been suggested that the defective supply of blood which passes into the aorta at each ventricular systole stimulates the heart to extra exertion, and thereby induces hypertrophy. A more rational explanation, however, of the hypertrophy (which, be it observed, is usually slight in this case) may be found in the fact: that when the pulmonary obstruction is considerable and produces engorgement of the systemic veins and general serous effusion, impediment to the circulation of the blood through the systemic capillary vessels necessarily results, and thus excites hypertrophy of the left ventricle. The hypertrophy of the left ventricle may be, in some cases, accounted for by co-existing disease of the aortic valves or of the aorta.

These alterations in the size of the different parts of the heart occasion deviations both from its natural position in the thorax and in its form. When, for example, the right ventricle is hypertrophied, the organ loses its natural conical shape, and becomes broader and rounder; it also assumes a more horizontal position, so that its apex is forced from its usual position towards the left lateral region of the thorax.

The hypertrophy causes an increase of the extent

of dulness on percussion over the præcordial region; and the position of the heart enables us to feel its impulse not at one point only, but for an inch or more along one intercostal space. The contractions of the hypertrophied ventricle also are perceptible at the *scrobiculus cordis*.

Diastolic Mitral Murmur is comparatively of rare occurrence. I have not met during the last eight or nine years with more than a dozen cases in which I could with certainty diagnose its existence. Some English authors ignore the possibility of such a murmur; and Dr. Latham speaks of it as a sort of clinical curiosity. On the Continent, on the other hand, authorities treat of the murmur as an established fact. This discrepancy in opinion is, doubtless, to be attributed to the rarity of its occurrence, and to the difficulty which usually attends its diagnosis. On account of the nature of the lesion which produces the murmur, viz. obstructive disease of the mitral orifice, the heart's movements are much deranged; its action becomes rapid and tumultuous, and its rhythm and the character of its sounds greatly altered.

In well-marked cases of diastolic mitral murmur, and where the heart's action is still vigorous, a loud and prolonged murmur is heard at the apex of the heart, and from thence upwards to the left of the nipple; this is often attended with a vibratile thrill —*frémissement cataire*—perceptible at the heart's

apex. The murmur is sometimes so prolonged as to occupy not only the whole natural period of the diastole, but also the interval of repose, and a part of the time of the systole of the heart. The impulse, in fact, appears to follow immediately upon the cessation of the murmur, or rather, to be its conclusion, and to wind it up; it is brief and rapidly accomplished, and, after a very short pause, is followed by the recurrence of the murmur.

The characters of a diastolic mitral murmur correspond and vary in their intensity with the degree of constriction of the mitral orifice, and with the power of the heart to carry on the circulation.

The heart is hypertrophied, the hypertrophy affecting, in a particular manner, its right side and its left auricle. The præcordial dulness is, therefore, found increased on percussion, especially in the direction of the right ventricle. The left ventricle may be of normal size.

In cases of this kind the aortic sounds and the beat of the arteries are often remarkably weak,—a fact which may be attributed to the small quantity of blood which passes through the constricted mitral orifice into the left ventricle, and which is therefore propelled into the aorta at each ventricular systole. In consequence of the weakness of the aortic sounds, the increased intensity of the second sound of the pulmonary artery becomes more than ordinarily remarkable.

Such are the physical signs which I have observed in cases of this affection. They are very characteristic of it;* and it is worthy of remark, that the general symptoms also correspond remarkably to the nature of the physical defect.

The breathing is difficult, and on the least exertion becomes laborious and hurried; hæmoptysis is of frequent occurrence, and readily excited; and rest

* As the account I have given of this murmur differs much from that of many stethoscopic authorities in this country, I have thought it well to show that it is backed by the observations of many authorities.

Corvisart speaks of it as,—

"Un bruissement particulier, difficile à décrire, sensible à la main appliquée sur la région précordiale, bruissement qui provient, sans doute, de la difficulté qu'éprouve le sang à passer par un orifice qui n'est plus proportionné à la quantité de fluide," &c.

"When the contraction of the mitral orifice is great," says Hamernjk, "the second murmur is long and loud; and some portions of it are louder than others, producing the hum of a spinning-wheel, and has thus been taken for a double sound; such a second (diastolic) murmur is protracted, and ends in the systole; there is a distinct pause between the systole and diastole of one complete heart's movements, but none between the diastolic and recurring systolic murmurs."

"It is especially," says Skoda, "in cases like these, that vibrations are felt when the hand is laid upon the præcordial region,—the *frémissement cataire* described by Laennec."

"It is a common opinion," says Professor Jacksch of Prague, "that a fremitus which is felt at the apex of the heart, and which accompanies the diastole, is a pathognomonic sign of an obstructive mitral orifice."

I may also refer to a paper on the "Diastolic Mitral Murmur," which I published in the *Edinburgh Monthly Journal* in 1854.

and quiet in an especial manner produce ease and comfort.

To arrive at a correct diagnosis, in cases of this kind, it is absolutely necessary *to fix the exact time of the heart's systole*, by feeling its impulse at the same time that its sounds are auscultated. The observer will thus be enabled to determine with which movement of the heart the murmer is synchronous—that is, provided its action is not excessively rapid, for in such case the most practised ear may be unable to unravel the complication of sounds heard.*

Of mitral valvular diseases, it may be said, generally, that they are more frequently met with than aortic valvular diseases in the earlier periods of life; and that in the great majority of cases they result from acute endocardial—rheumatic—affections. Bamberger tells us, that of 230 cases of valvular diseases, 150 were mitral; and that of these 63 were found in men, and 87 in females. When mitral valvular diseases occur in advanced life, they are usually the consequences of calcareous and atheromatous degenerations of the valves.

* I may observe, that in no kind of valvular diseases does the action of the heart seem more amenable to the influence of digitalis than in this. Consequently, in these cases this medicine affords us an excellent means of analysing the sounds and murmurs of the heart by quieting its action. Bamberger, in his recent work on the Heart (p. 251), corroborates this statement; he speaks of the remedy as being invaluable in this condition of the heart.

Aortic Valvular Murmurs.—I have already described the pathological changes of structure to which the aortic valves are liable. The consequences which may result from these changes, are: 1st, obstruction to the free flow of the blood out of the ventricle; 2nd, its regurgitation into the ventricle; or 3rd, both obstruction and regurgitation. A systolic murmur is produced in the first case; a diastolic murmur in the second; and a double murmur in the third. Fibrinous coagula, which have formed in the heart during life, and when the valves and the aortic orifice is perfectly healthy, are also supposed occasionally to excite systolic murmurs.

A *Systolic Aortic Murmur* is heard loudest over the site of the aortic valves; that is, over the middle of the sternum, about opposite to the third intercostal spaces; it passes thence in a direction upwards in the course of the current of the blood, and may often be distinctly heard in the cervical arteries. It is not always audible at the apex of the heart. The second sound over the left ventricle usually becomes indistinct in cases where there exists a systolic aortic murmur; and the reason of this is, that the state of the valves which produces the systolic murmur interferes with their proper action.

When the aortic murmur is heard louder towards the top of the sternum than over the aortic valves, it is probable that the disease provoking it lies rather in the aorta itself than at its valvular orifice.

But the absence of a murmur over the aorta, during the heart's systole, does not necessarily indicate a healthy condition of the aorta; if the first sound be indistinct, or duller than natural, and the heart's impulse of ordinary strength, we may conclude that the coats of the aorta are not in a normal condition.

Hypertrophy and dilatation of the left ventricle are the necessary consequences of constricted aortic orifice, and of disease of the aorta. Their degree depends on the degree of obstruction which is offered to the circulation, on the condition of the muscular tissue, and on the age of the disease.*

Aortic Regurgitant Murmur indicates a defective condition of the aortic valves, permitting the blood to regurgitate from the aorta into the left ventricle during the ventricular diastole. Its point of greatest intensity is over the aortic valves; that is, about the middle of the sternum, and opposite the third intercostal spaces; from thence it is carried downwards, *i. e.* in an opposite direction to a systolic aortic bruit. This diastolic murmur is often heard very loud over the lower part of the sternum, and appears, generally speaking, to be conducted downwards along the

* When we are considering the physical signs and symptoms of valvular diseases, it is requisite in all cases, as far as possible, to take *the age* of the disease into our calculation. Hypertrophy requires time for its production; whilst the injury which occasions the murmur may be effected in a few days.

sternum—that is, in the direction of the right ventricle, rather than in that of the left ventricle, which is contrary to what is usually supposed to happen. I have, indeed, often heard a loud regurgitant aortic murmur at the lower part of the sternum, when the second sound (probably that of the pulmonary artery's valves) was clearly audible at the apex of the heart, and unmixed with any murmur. The reason why the regurgitant current so often conveys the diastolic murmur downwards along the sternum rather than towards the apex of the left ventricle is not clear.

The first aortic sound may be healthy in cases of diastolic aortic murmur: but in the majority of cases it is altered, because, when the valves are so disordered as to permit of regurgitation, they almost always at the same time present some obstruction to or produce some alteration in the current of blood which flows through them during the heart's systole, such as will occasion a murmur.

The state of the pulse in aortic valvular regurgitation is peculiar. The artery during systole is suddenly, and when the left ventricle is hypertrophied is forcibly, filled with blood, but during diastole it abruptly collapses under the finger. The phenomenon depends upon the defective condition of the aortic valves, which no longer duly sustain the columns of blood during the ventricular diastole. When the aortic valves are defective, there is, also,

an absence of the healthy second sound usually heard in the cervical arteries; or this second sound is replaced by the diastolic murmur.*

The second aortic sound is often heard remarkably loud, and even ringing over the ascending aorta; the first aortic sound being at the same time unnaturally feeble. This condition of the sounds generally indicates the existence of a calcareous condition of the aorta; the aortic valves being still healthy, and capable of closing the aortic orifice. This increase of the second sound is supposed to result from the hardened state of the arterial walls, rendering them better reflectors of sound.

A double Aortic Murmur, systolic and diastolic, frequently occurs as a consequence of disease of the aortic valves. In such cases, the systolic murmur is, as a rule, heard loudest in a direction upwards from the situation of the aortic valves, and the diastolic murmur in a direction downwards.

Aortic valvular diseases are less frequently observed, as the results of acute rheumatism, than mitral valvular diseases. They are more often met with at advanced periods of life, and as consequences of calcareous and atheromatous degeneration of the structures of the valves. The male sex, moreover,

* This absence of the second sound in the cervical arteries in cases of defective aortic valves is, it may be incidentally observed, a proof that the second cardiac sound depends upon the tension of the aortic valves during diastole.

appears to be much more frequently than the female subject to them,—a fact contrary to what appears to hold good in the case of mitral diseases.

Hypertrophy and dilatation of the left venticle of the heart are the immediate consequences resulting from aortic valvular diseases and from diseases of the aorta. When the valvular disease is of some standing, the hypertrophy of the left ventricle is often very great; and then the impulse of the heart may be felt widely over the præcordial region. The impulse has a peculiar heaving character (often visible at a distance): in such case, during diastole, there is a marked falling backwards of the thoracic walls. The apex of the heart is also felt more towards the left, and sometimes much lower down than natural. The præcordial dull percussion also is increased in extent, and rather towards the left and downwards, than towards the sternum, as occurs in hypertrophy of the right ventricle. The cervical arteries are also enlarged, and beat strongly at each impulse of the heart.

The hypertrophy of the left ventricle of the heart, in fact, is more marked in aortic than in any other valvular disease; but there can be no doubt, that in many cases the diseased condition of the aorta itself, and of the arteries generally in the body, assists in producing it. The structure of the aorta is, in fact, frequently more or less altered when the aortic valves have undergone atheromatous degeneration. Its coats are hardened and thickened by

the deposits and their elasticity is thereby much impaired, and an unnatural impediment to the circulation established.

Tricuspid Valvular Murmurs. — Murmurs arise much less frequently in the right than they do in the left cavities of the heart—a fact which accords with what has been already said of the pathology of valvular diseases. The tricuspid valves are rarely primitively affected by endocarditis or atheromatous disease. They are much more frequently affected secondarily to the valves of the left side of the heart, and seldom to such an extent as to render them incapable of performing their office. Systolic murmurs, however, are occasionally heard over the right side of the heart; and there is no doubt that, in such case, they sometimes result from the circumstance of the valves being incapable of closing the tricuspid orifice, in consequence of inordinate distension of the ventricle. The right ventricle will naturally dilate much more readily than the left under an equal degree of pressure, on account of the comparative thinness of its walls.

When tricuspid regurgitation takes place to any great extent, the blood accumulates in the right auricle, and the venæ cavæ; and may so distend the large veins as to render their valves incompetent, and consequently to occasion visible pulsations in the external jugular veins. It is certain, however, that regurgitation often takes place through the tricuspid

orifice, without producing a murmur. The diseased conditions which produce the murmur in the case of the mitral valves are very rarely present here, and yet tricuspid regurgitation is very common. It must also be remembered, in judging of murmurs heard over the situation of the right side of the heart, that they may arise at the mitral or aortic orifices and be conducted from thence; and that diseases of these orifices are apt to produce that very condition of the right ventricle, viz. its dilatation, which is supposed to occasion a systolic tricuspid murmur. Hence, the coexistence of a murmur over the right side of the heart, and distension of the jugular veins, do not of necessity indicate disease of the tricuspid valves.

Fibrinous coagula, also, formed during life in the right side of the heart, will occasion murmurs over the right side of the heart. Of this fact I have elsewhere given an interesting example, the subject of the disease being a well-known member of our profession. The dilatation of the right ventricle and auricle, which coexists with tricuspid regurgitation, occasions an increase of the dull percussion-sound over the lower part and towards the right side of the sternum. The distended cavities sometimes reach to some distance beyond the right border of the sternum. The murmur (if really tricuspid) will be heard loudest at the lower part of the sternum; and it has generally the character of a smooth, not

over loud, blowing murmur. We may be sure that the murmur arises in the right ventricle when no murmur is heard at the same time over the mitral or tricuspid orifices. A cardiac murmur, also, heard louder at the right lower border of the sternum than over the left and base of the heart, may also surely be considered as of tricuspid origin.

Murmurs, systolic or diastolic, resulting from structural disease of the *pulmonary artery* or its valves, are very rarely met with.

The following symptoms have been observed in a case of exceeding constriction of the orifice of the pulmonary artery:—Loud, prolonged, systolic murmur; loudest in the third intercostal space, close to the sternum, and heard along the lower border of the third left rib for the space of one to two inches; the murmur being inaudible about the top of the sternum and its right upper border; slightly audible over the aortic valves, and downwards over the right ventricle; the first sound being replaced by the murmur at the base of the heart, but remaining audible at the apex.

Systolic murmurs, however, (not of diseased structural origin,) are frequently heard over, and seem to proceed from, the pulmonary artery; but the causes of such murmurs are obscure. They in all probability result either from altered condition of the blood or of the contractibility of the artery, or from the mechanical influence of pressure acting from

without upon the artery.* I believe that they are also not unfrequently produced by anæmia. Murmurs in the pulmonary artery are also heard in certain cases of tubercular disease of the apex of the left lung. I have already referred to the opinion, that systolic murmur heard in the left clavicular region may be in some cases caused by pressure of the hardened lung upon the subclavian artery. I am inclined to think, that one of the characteristics of a systolic murmur of the pulmonary artery is the very limited space of the thoracic walls over which the murmur is audible — a fact which might, indeed, be anticipated from its anatomical distribution—and that by this sign we may distinguish it from the more widely diffused murmur, which arises in the subclavian artery.

* I once noted a loud, persistent bruit over the pulmonary artery in a case, in which, after death, one of the lobes of a very enlarged thymus gland was found lapping over the root of the artery.

CHAPTER XIV.

HYPERTROPHY OF THE HEART.

PATHOLOGY.—NATURAL WEIGHT, SIZE, ETC., OF THE HEART.—HYPERTROPHY. — ITS CAUSES. — HYPERTROPHY OF THE LEFT SIDE OF THE HEART.—HYPERTROPHY OF THE RIGHT SIDE OF THE HEART.—DILATATION OF THE HEART.—ACTIVE, SIMPLE, PASSIVE DILATATION. — CAUSES OF DILATATION. — SIGNS OF HYPERTROPHY.—ENLARGEMENT OF THE THYROID GLAND WITH PALPITATIONS, ETC.

By hypertrophy of the heart is understood an increase of its muscular tissue—the muscular tissue, thus increased, being either perfectly healthy, or more or less degenerated.*

Hypertrophy may be partial, affecting one side of the heart—one ventricle or one auricle only; more generally, however, it affects several portions of the organ at the same time; and it is in most cases associated with dilatation of the heart's cavities.

In order to arrive at an estimate of what may be properly called hypertrophy of the heart, it is

* It has not yet been satisfactorily determined whether the muscular fibres increase in size in hypertrophy of the heart; but there is no doubt that they increase in number.

necessary that we should possess some measure by which to judge of its size and weight in health. In fixing such a standard, however, we must be contented with something approximating to the truth; for the relative weight of the heart to the body in healthy individuals differs much.

From calculations which have been made on this subject, it appears that the medium weight of a healthy adult heart ranges between eight and ten ounces, and that the weight increases with age—at least, in the male.

The measurements given by Bizot, of the average thickness of the walls of the heart's ventricles, are as follows :*—

	English inches. In men.	In women.
Thickness of the walls of the left ventricle at the base	0·43	0·36
Ditto at the middle	0·45	0·39
Ditto near the apex	0·31	0·28
Thickness of the septum of the ventricles at the middle	0·43	0·39
Thickness of the walls of the right ventricle at the base	0·16	0·15
Ditto at the middle	0·11	0·11
Ditto near the apex	0·08	0·08

* These measurements of Bizot must be considered more accurate than those given by Bouillaud. I have taken them as reduced in Dr. Stokes' work to English inches. The following are Bouillaud's measurements :—

Thickness at base of left ventricle	6 to 7 lines.
,, ,, right ventricle	2½ ,,
,, ,, left auricle	1½ ,,
,, ,, right auricle	1 line.

And these are his measurements of the size of the valvular openings of the heart :—

	English inches. In men.	In women.
Width of the left auriculo-ventricular orifice	4·29 ..	3·61
Ditto of the right	4·81 ..	4·18
Width of the origin of the aorta above the valves.....	2·74 ..	2·49
Ditto of the pulmonary artery	2·79 ..	2·60

The cavities of the right side of the heart have also, as it would seem, a greater capacity than those of the left side; and the auricles a capacity less than that of the ventricles.

The above measurements may be taken as representing, with tolerable accuracy, the thickness of the walls of the heart when its cavities are distended to their natural size—a condition which it is important to bear in mind whilst considering the subject of hypertrophy, for the reason, that when the healthy heart is much contracted its walls necessarily become thicker than natural; that is, than the standard measurements above given of them. The very contracted state of the heart which is sometimes met with after death, and to which the name of concentric hypertrophy has been applied, is in reality a simple thickening of the ventricles, resulting from inordinate contraction of the heart, and consequent diminution of its cavities. This condition of the heart is observed occasionally in anæmia and phthisis, and invariably, according to Cruveilhier, in the bodies of persons guillotined.

On the other hand, again, when its walls are of their natural thickness, and its cavities dilated, the heart—according to the definition of hypertrophy above given—must be considered as hypertrophied, inasmuch as the bulk of its muscular tissue—and consequently its weight—is abnormally increased.

The most satisfactory mode, therefore, of judging of the hypertrophy of a heart, is by a consideration of its weight, as well as of the measurements of its parts.*

* The following observations on the weight of the heart in health and in disease are taken from a very instructive paper by Dr. Peacock (*Edin. Monthly Journ.*, 1854):—

"The average weight of the healthy heart in males, between 20 and 55 years of age, is 9oz. 8dr.; that in females of like age, 8oz. 13dr. The calculations, however, are arbitrary, for it is not easy to say at what size a heart ceases to be healthy.

"The average weight of the healthy heart in those who die of chronic and wasting disease is less than in those who die after a short illness. In cancer of the stomach, and chronic affections of the liver, the heart will be found to weigh occasionally only five or six ounces; and in large men who have died suddenly, it may be twelve ounces. The heart increases in weight with advance of life; but, perhaps, in very advanced age undergoes a decrease.

"In phthisis the weight is less than natural, but its wasting is counteracted by the impeded respiration causing its hypertrophy. In chronic bronchitis it ordinarily acquires a considerable size; and in diseased kidney there is a tendency to enlargement.

"In twenty-four cases the heart weighed more than twenty ounces; and in all these, except two, there was present marked aortic or some other valvular disease. In one of the two, the heart weighing twenty ounces, there was slight atheromatous disease of aortic and mitral valves, and of the aorta; and in the

The following are the kinds of hypertrophy of the heart usually met with :—

1. *Simple hypertrophy*, which consists in a thickening of the walls of the heart, the cavities remaining of their natural size.

2. *Eccentric hypertrophy*, consisting in an enlargement both of the cavities and of the walls of the heart.

When the hypertrophy is uncomplicated, it consists simply in an increase of healthy muscular fibres. But the hypertrophied tissue is sometimes partially degenerated, and then it presents, at those parts, the appearances characteristic of the particular degeneration which it has undergone. The proper walls of the ventricles, and the columnæ carneæ and papillary muscles, are generally all enlarged together, but sometimes one of these parts more so than the other. The complicated columnæ of the right ventricle are often remarkably increased, and out of proportion to the other parts of the walls of the cavity; they are so large, prominent, and numerous, as sometimes almost to separate the cavity into two or many divisions. The nervous ganglia and the coronary arteries, also, increase in size at the same time.*

other, weighing forty ounces, chiefly hypertrophy and dilatation of right ventricle: there was no valvular disease, and no cause of death given."

* This has been shown by Dr. Lee in his beautiful monogram on the nerves of the heart.

The ventricles are more frequently hypertrophied than the auricles, and the left ventricle more frequently than the right. Dilatation of the cavities of the heart is usually associated with hypertrophy of its walls: for the cause which induces the latter condition, naturally promotes the former likewise. Dilatation with hypertrophy of both the ventricles, is the condition of the heart most commonly met with in cases of advanced hypertrophy; the reason of this being, that the obstruction of the aortic orifice, for instance, which at first excited increase only in one ventricle, the left, at length excites, indirectly through the lungs, increase in the right ventricle. With regard to the auricles, it may be observed that their hypertrophy, unless when well marked, is readily overlooked, and not easily defined; their exact natural measurements having not yet been satisfactorily ascertained. The extent to which hypertrophy of the heart may reach in man can be gathered from this,—that the left ventricle has been known to have acquired a thickness of two inches, and the heart itself to weigh as much as five pounds.

When the hypertrophy of the heart is considerable, both the natural form of the organ, and its position in the thorax, are altered. It loses its usual conical shape, taking a more globular, and sometimes even a squarish figure, so as to become broader than it is long; its ordinary projecting

apex at the same time disappears. The position of the heart also is changed: it lies more transversely than natural, its apex reaching away towards the left lateral region of the thorax; so that its beat, instead of being felt to the right of the left nipple, is felt to the left of it, and if the hypertrophy be great, as low, or even lower than the sixth rib. The enlargement of the heart, however, is most marked in the situation of that portion of the organ,—auricle or ventricle,—which is most particularly hypertrophied.

Hypertrophy of the heart may, practically speaking, be looked upon as a condition resulting from the presence—either within or without the heart—of some hindrance to its freedom of action; the hindrance being such as the natural-sized organ has not force enough to strive against successfully for any long time,—that is, so as duly to supply the wants of the system. To overcome the impediment, the heart is, in the first instance, stimulated to extra exertion, and then, according to the beneficent principle, *ubi stimulus ibi fluxus*, the stimulus provokes an increased supply of nutritive materials, and so the muscular tissue is multiplied and strengthened. Here, as in the homely instance of the blacksmith's arm, the increased demand for force gives birth to an increase of the nutritive supply which generates the force.

Hypertrophy of the heart must therefore be re-

garded rather as the result of an effort of nature, striving to compensate for the defective condition of other parts, than as a disease tending to the destruction of life. The time has been, when the whole efforts of his art were employed by the physician in the vain attempt to reduce this necessary hypertrophy, which nature had provided for an especial occasion.

The existence of such a disease as simple hypertrophy is very doubtful. As a rule, the hypertrophy may be traced to a distinct cause; and when we in any instance fail so to trace it, we may ascribe the failure, not to the non-existence of a cause, but rather to our inability to discover it.

Causes of Hypertrophy.—The above remarks naturally lead us to consider the diseased conditions which excite the hypertrophy, or, in other words, its causes. These we shall find to exist both within the heart itself, and in parts external to, and even distant from, the organ. They consist, for the most part, in mechanical impediments to the circulation of the blood. In some few cases the hypertrophy results from causes which we are not able clearly to trace out.

The chief and most ordinary causes of hypertrophy of the heart, are found in the organ itself, and in the great vessels which arise from it. They consist in diseases of its valves, and in abnormal states of its orifices and its great vessels. Defective

aortic valves, permitting regurgitation of the blood into the left ventricle during its diastole; constriction of the aortic orifice, impeding the free passage of the blood from the left ventricle during its systole; deficiency of the aortic valves, associated with constriction of the aortic orifice; defective mitral valves, permitting regurgitation of the blood from the left ventricle into the left auricle;—all these abnormal conditions occasion impediments to the circulation of the blood through the heart, and their immediate effects are, for the most part, communicated directly to the left side, and, indirectly, to the right side of the heart.

I have already said that the left ventricle is the part of the heart most frequently hypertrophied, and this accords with the fact that diseases of the valves (such diseases, at least, as render them defective) are frequent on the left, and comparatively rare on the right side of the heart.

But notwithstanding that the valves of the right side of the heart are rarely thus diseased, we very often find the right ventricle hypertrophied and dilated. The chief causes of this are the impediment to the pulmonary circulation which arises from chronic diseases of the lungs, or from defects of the valves of the left side of the heart. Their action is exerted indirectly in the following manner. The disease of the mitral or aortic valves hinders the free passage of the blood from the lungs into the

left side of the heart, and thus obstructs the pulmonary circulation ; the consequence of this is, that the blood is thrown back upon the pulmonary artery and right ventricle, and the right ventricle stimulated to extra exertion, in order to overcome the obstruction. And thus are occasioned its hypertrophy and dilatation.

After the same manner we may trace back, even still further along the current of the circulation, the consequences of valvular diseases of the left side of the heart ; we may thus see how a hindrance to the passage of the blood through the aortic orifice at length communicates its effects, even to the systemic capillary circulation at the most remote parts of the body. The impediment to the blood arising from aortic valvular disease, which we have already traced back as far as the right ventricle, is readily communicated to the blood passing from the right auricle, and from this again to the venæ cavæ, and so backwards along the venous system, even to the capillary circulation. In consequence of the capillary circulation being obstructed, the blood does not flow readily along through the arteries ; and fresh force is required to drive it on. The heart is, therefore, called upon to supply the required extra force ; and hence we have another cause of hypertrophy of the left ventricle, in addition to the obstruction at its aortic orifice. This new obstruction is secondary to and the consequence of the first.

Mechanical impediments to the passage of the blood through the aorta and large arterial trunks are also causes, external to the heart, which give rise to hypertrophy and dilatation; and their effects are the greater the nearer they are situated to the heart. They may result from contraction of the arterial trunks, whatever its cause, whether pressure from without, or disease within; from aneurisms, and dilatations, and atheromatous degenerations of their coats, whereby their natural elasticity and contractility is destroyed.

An atheromatous state of the smaller arteries, also, is not an unfrequent cause of hypertrophy of the left ventricle. I believe that such a state of these vessels existed in many of the recorded cases of hypertrophy of the left ventricle, in which it is said, that there existed no physical cause of the hypertrophy. And it is worthy of remark, that the degree of atheroma existing in the aorta is (according to my experience at least) far from being a measure of the amount of atheromatous degeneration in the smaller arteries. I have frequently seen the smaller arteries much diseased when the coats of the aorta were only slightly affected, and have recorded cases of this kind in the *Pathological Society's Transactions*. The following is an example :—

In a man who died suddenly from apoplexy, I found the heart to weigh twenty-four ounces. Its muscular tissue was firm and healthy, its valves were

competent, and all other parts of the body, excepting the brain and the blood-vessels, healthy in appearance. There was a large clot of blood in the brain, and the cerebral vessels were very atheromatous. Now, as it is probable that the condition of the cerebral vessels was the condition of the smaller arteries in all parts of the body, I conclude, that the obstruction to the circulation of the blood thereby produced was the cause of the cardiac hypertrophy.*

Impediments, moreover, to the pulmonary capillary circulation, give occasion to hypertrophy and dilatation of the heart. Whatever, in fact, interferes with the pulmonary circulation acts directly as a cause of active dilatation and hypertrophy of the right ventricle. Thus all diseases of the lungs, which suddenly obstruct the pulmonary circulation by destroying, or rendering useless for a time, a large amount of lung-tissue,—emphysema, enlargement of the bronchi, certain deformities of the thorax, pleuritic effusions, and pneumonia,—all these must be considered as sources of hypertrophy and dilatation of the right side of the heart. But no cause acts more effectually, through the lungs, in inducing hypertrophy and dilatation of the right side of the heart than constriction of the mitral orifice.†

* It has been demonstrated that the force required to drive fluids along tubes with solid walls, *when the force which moves them is intermittent* (as in the heart), is much greater than that required to drive them through elastic tubes, such as the healthy arteries.

† I have met with a case of great hypertrophy of the right

In cases of uræmia, where hypertrophy of the left ventricle exists without disease of the valves or of the arteries, the hypertrophy must be attributed to the impediment to the systemic capillary circulation, and the œdema, which results from the uræmic condition of the blood.

Dilatation of the heart's cavities is associated with different conditions of its walls:

1. With hypertrophy of the walls.
2. With normal thickness of the walls.
3. With thinning of the walls.

The first condition, *active dilatation*, is that most commonly met with. It is, indeed, merely one form of hypertrophy,—excentric hypertrophy,—but it is placed under the head of dilatation, because the dilatation predominates over the hypertrophy. The auricles often become the seat of active dilatation, especially when the auriculo-ventricular orifices are much constricted.

The second condition is *simple dilatation*, the cavities of the heart being dilated, and their walls of normal thickness. Here, however, although the walls of the cavities are of normal thickness, the bulk of the heart's muscular tissue is increased, and the heart, therefore, hypertrophied.

side of the heart, in which there existed a considerable amount of atheromatous degeneration of the branches of the pulmonary artery. In this case there was no valvular disease on the left side of the heart, nor other cause (besides the one mentioned) to which the hypertrophy could be ascribed.

The third condition is *passive dilatation*, the walls being relaxed and attenuated, and the cavity of the heart enlarged. It is the only form of dilatation which demands our attention here, for the other forms of dilatation come more properly under the head of hypertrophy.

In passive dilatation the muscular tissue of the heart is softer and weaker and thinner than natural, and is often found to have undergone partial degeneration. The walls of the heart collapse when cut across, instead of standing out firm as in health. This enfeebled state of the heart is the result either of the degeneration of its tissues, or of wasting of the muscular tissue; and may occur as a consequence of pericardial or endocardial inflammation, or in connexion with wasting and constitutional diseases.

We may readily understand why passive dilatation should occur when the heart is thus weakened; for if, under such circumstances, any impediment to the performance of its functions arises, the muscular walls of its cavities, instead of overcoming, yield to the extra pressure, and dilate. Had the tissue been healthy, hypertrophy would naturally have resulted from the impediment.

Passive dilatation generally affects both ventricles together; the left ventricle is sometimes reduced to a third of its natural thickness, and at some points, particularly about the apex of the heart, its walls are occasionally so attenuated as to be diaphanous.

But the right ventricle is more liable to extreme thinning than the left. In dilatation the heart is enlarged in its transverse direction, and assumes a peculiar globular appearance.

The orifices of the heart frequently enlarge at the same time that its cavities dilate; and the valves themselves, and their tendinous attachments, are likewise enlarged, and are consequently enabled still duly to perform their functions.*

The exciting causes of dilatation of the heart are, for the most part, the same as those which occasion hypertrophy of the heart; what has been said, therefore, concerning the causes of hypertrophy, applies generally here.

Hypertrophy and Dilatation of the Heart: Physical Signs.—The local physical signs which indicate the existence of hypertrophy and dilatation of the heart consist in alterations: 1st. Of its healthy sounds, in respect of loudness, clearness, and duration; 2ndly. Of the natural force and character of the heart's impulse, and of the situation where, and of the extent

* I am inclined to think that the condition of the coronary arteries, especially of their aortic mouths, influences much the form which the heart assumes in valvular and other diseases of it. When, for instance, the mouths of the coronary arteries are contracted—a fact of frequent occurrence—as a consequence of atheromatous or fibroid disease of the aorta, or when the coats of the branches of the coronary artery are much degenerated, the muscles of the heart are improperly nourished—the arterial supply being necessarily defective—and, consequently, dilatation is more likely to result than hypertrophy.

of surface over which it is perceptible; and 3rdly. Of natural extent of dulness produced by percussion over the præcordial region.

Hypertrophy and dilatation of the heart increase the extent of præcordial dull percussion-sound, and in proportion to their degree. When one portion only of the heart is thus enlarged, the dulness increases especially over the situation of that part—across the lower part of the sternum, for instance, when the right side of the heart is enlarged; and towards the left side of the thorax when its left side is enlarged. When the dilatation exceeds the hypertrophy, the dulness increases chiefly in the transverse direction; and when the reverse of this occurs, it increases also in a direction from above downwards, —that is, in the long diameter of the heart. In health, the dulness is not perceived over a surface of more than about an inch and a half of the præcordia, but it may, when the hypertrophy is considerable, reach over an area two or three times greater. The dulness arising from hypertrophy and dilatation of the heart, may be distinguished from that which results from pericardial effusions: by an examination of the history of the case; by the attendant symptoms; and by the fact, that the dulness of hypertrophy increases in a direction downwards and towards the left side, and the dulness arising from effusion, upwards. The resistance felt on percussion also is much more marked in pericardial effusion than in hypertrophy.

It must not be forgotten, while considering these signs, that pneumonic and tubercular infiltrations into those portions of the lungs which immediately surround the heart, may render the præcordial percussion-sound abnormally dull; and, on the other hand, that the dull percussion over an hypertrophied heart may be diminished by emphysema of the lungs, which causes their borders to overlap the heart to an abnormal degree,—a condition, indeed, which is not unfrequently associated with enlargement of the right side of the heart. Under such circumstances, the amount of dulness, which is natural to hypertrophy, may be actually diminished. The same results may ensue when the thoracic cavity is rendered larger and wider than natural, so as to allow great expansion of the lungs.

Percussion does not allow us to distinguish between hypertrophy and dilatation of the heart; both states give a like percussion-sound.

CHAPTER XV.

TREATMENT OF VALVULAR DISEASES.

TREATMENT OF ENDOCARDITIS.—GENERAL.—LOCAL.—TREATMENT OF CHRONIC VALVULAR DISEASES.—PULMONARY DISORDERS.—ABDOMINAL.—GENERAL TREATMENT.

THE treatment of acute valvular diseases, which necessarily comprises that of acute endocarditis, is to be conducted on the same principles as the treatment of acute pericarditis. The near relation of these diseases has been already spoken of; their frequent coexistence; their origin from, or connexion with, the same exciting cause; and the anatomical similarity of the inflamed structures. Acute endocarditis, indeed, rarely comes under our observation except in connexion with acute rheumatism.

The dangers, however, to be anticipated from the results of endocarditis, are greater even than those which belong to pericardial inflammation. The pericardial membrane may receive a permanent injury; lymph may be deposited upon it, and partial adhesions of its surfaces may result, and yet no

damage be done to the general working of the heart. The inflammation may pass away, and the organ still be left capable of performing its duties. Not so is it with the endocardium; the force of the inflammation falls upon the valves, whose smallest injury is of the most serious importance; and a few hours of the inflammatory action may ruin them for ever.

Hence it follows, that in acute endocardial disease that treatment only can be considered as perfectly efficacious, which anticipates and wards off the development of the mischief which we dread. Unfortunately, however, it happens that the first certain proof we have of the existence of valvular disease, is the substitution of unnatural murmurs for the ordinary healthy sounds of the heart; and these murmurs tell us that the inflammation has already reached beyond the stage of congestion, and has made progress in its work of disorganization; that our remedies now come late into the field to combat the malady; and that the valve may be already irretrievably damaged, beyond the *vis medicatrix naturæ*, or the art of medicine to repair.*

It follows from this, that we should endeavour, by a most careful watching of signs and symptoms, in

* There is no doubt that endocardial murmurs which have arisen during the acute period of endocarditis, do, occasionally, disappear with the inflammation; but in the very great majority of cases, it is certain that the murmurs become permanently established.

all those affections during whose progress we observe endocarditis frequently to arise, to be beforehand, as it were, with the inflammation,—to be busily employed in the treatment of the valvular disease, or rather of the diseased condition of the body which occasions the valvular disease, before this final test,—the valvular murmur,—gives us the proof of its existence. And certainly, in the majority of cases, there are signs from which we may obtain the knowledge that injury to the valves is imminent; and obtain it, indeed, with such an amount of assurance as to justify us in acting as if the positive proofs—namely, the valvular murmurs—had demonstrated the existence of the endocarditis. When, for instance, during an attack of acute rheumatism, we find the heart's action become suddenly violent and irregular; its sounds altered in character, though still to be called healthy sounds; præcordial pain, or tenderness on pressure over the heart; high fever, and irregular pulse, there can be no need for us to wait for a cardiac bruit to decide our curative proceedings. To anticipate thus the endocardial inflammation in its early aggression, becomes the proper object of the skill of the physician turned to its highest purposes—the prevention of disease. Unfortunately, he possesses no remedy which will enable him to cut the inflammation short, whenever it has once fixed itself in the endocardium.

For the general—the constitutional—treatment of

rheumatic endocarditis, I may refer the reader to what has been said of the general treatment of rheumatic pericarditis. It consists, mainly, in the endeavour of neutralising or eliminating the rheumatic element from the body—in curing the rheumatic fever. Thereby we can alone hope to anticipate the occurrence of the endocarditis, or to arrest its progress when it has once made its existence manifest.

Those two prime remedies, bleeding and mercury, once thought so powerfully efficacious in arresting inflammation, have been generally condemned by modern experience as useless and inexpedient, and are not now resorted to for that object. We cannot, indeed, expect to remove the cardiac inflammation by the use of any violent medicinal agencies specially directed to it.

But that both bleeding and mercury are remedies often of great utility in the course of the inflammation, I do not doubt for a moment—venesection, judiciously practised for the special object referred to under the head of pericarditis, viz. relief of any accompanying pulmonary and cardiac congestions; and mercury used as a promoter of the healthy secretions of the different organs of the body. I have already insisted upon the great service rendered by moderate bleedings, in congestions of the heart and lungs, in whatever way the congestions may have been produced; and have no reason whatever to doubt their utility when practised in the congestions which

arise, incidentally, during the progress of endocarditis.

As regards local treatment—leeches, cupping, &c., over the præcordial region—it may be observed: that experience does not justify the belief that it is as effective in endocarditis as we know it to be in pericarditis. How, we may well reason, can leeches or blisters applied to the surface of the thorax affect the inflammatory process which is going on inside the heart? Their effects on the pericardial inflammation is, as I have already shown, readily explained. When these local remedies, then, are of service—and I admit that they often are of service in endocarditis—I consider that they act beneficially, not directly upon the endocarditis but upon those other inflammations, viz. the pericarditis and pleurisy, which are frequently associated with it.

The treatment here recommended I consider the rational, and therefore, in the present state of our knowledge, the true treatment of endocarditis; and the results of the practice founded on it tend to show, that by thus actively combating the rheumatic fever, we employ the most effectual means which we possess of preventing coincident cardiac affections. But we must never be too sanguine of success, for our best applied resources often fail us here. Despite of all efforts, the inflammation will frequently attack the heart, and effect a permanent injury upon it.

Considerable difficulty sometimes attends the treat-

ment of secondary attacks of endocarditis; for it is not always easy to determine whether the endocarditis is the result of former inflammation of the heart, or whether it is the result of inflammation at present in action. We have no longer that sign, which was originally pathognomonic (inasmuch as it arose under our immediate observation) of the endocarditis, to help us, namely, the cardiac bruit; for this already existed, demonstrative of previous valvular injury. We must, therefore, in such a case, judge of the presence or absence of local inflammation, and of its degree, by the presence or absence of general febrile symptoms, of arthritic affections, and of all those other symptoms, which have been described as indicative of acute endocardial inflammation, and also by the state of the heart.

If, taking into consideration the previous history of the patient, we find, that he has suffered from former attacks of rheumatic fever, and been more or less ailing since the attacks,—subject to occasional cough, shortness of breath, and palpitations; and if, in addition to the cardiac bruit, there exist signs of hypertrophy of the heart,—then, in such case, and if there be no febrile symptoms present, we may conclude: that the valvular lesion is not of recent date.

If the evidence leads us to consider the inflammation as recent, we must proceed in our treatment as in an ordinary case of acute valvular disease; but

with this difference,—that the particular condition of the patient must be carefully taken into calculation, that is, the fact of his heart having been already organically diseased, in consequence of which the different functions of the body may have become more or less disturbed and weakened. But no rules for application of the treatment under such circumstances can be laid down; each individual case must be judged of by its own particular characters.

Chronic Valvular Diseases: Treatment.—We now come to consider the treatment of chronic valvular diseases, or rather, of the secondary disorders which necessarily result from them. And under this head we may comprise all those diseased conditions of the valves which impair their function, and are unconnected with inflammatory action—both those which result from the acute endocardial inflammation, as well as those also which are occasioned by slow degenerative changes of structure.

When the fact of a valvular imperfection is established by its proper diagnostic signs, and the endocardial inflammation which attended its production has passed away, the local injury has reached a condition over which our art has no direct control. The pulmonary, the abdominal, and the cerebral disorders, to which the valvular disease gives rise, are those which must claim our attention.

The treatment of chronic valvular diseases involves two especial indications, namely: first, the pro-

phylactic means, by which we endeavour to guard against a recurrence of the endocardial inflammation; and, secondly, the alleviation or removal of the accidental disorders which arise as consequences of the valvular disease.

The chief particulars to be attended to as prophylactic measures are the following:—Abstinence from whatever excites the heart's action inordinately; careful attention to the diet and clothing; avoiding exposure to sudden changes of temperature, to wet and cold, and all those influences which render the body liable to rheumatic seizures; moderate exercise; and the due maintenance of the excretory functions of the body.

Few, however, of those affected by heart disease are able to subject themselves to such favourable conditions. The bulk of humanity must labour and toil; and daily labour and toil necessarily entail exposure to influences hurtful to the disordered body; and thus it is that, in the great majority of instances of cardiac affections, the progress of the disease is continual, and the severity and complication of the symptoms ever on the increase.

Where the condition in life of the patient is such as to enable him to take advantage of all proper hygienic aids, and when, at the same time, the valvular disease has not reached beyond a certain degree, a long life may be often passed in freedom from most of the ordinary consequences of such

disease. Loud and persistent bruits may thus indicate organic disease of the heart, and yet only slight symptoms be present to enforce the existence of the disease on the minds of the patients themselves. The fact is encouraging for treatment; but without treatment—that is, proper prophylactic treatment—we cannot hope that such a favourable condition of things can long exist. The very fact of the existence of the bruit indicates the constant liability, in which the subject of it stands to a recurrence of the inflammation, if the injury be of inflammatory origin, and to a constant increment of the degeneration if it depend upon other degenerative changes of structure.

In all cases, therefore, whenever the stethoscopic signs leave no room for doubt concerning the existence of heart disease, it is the duty of the physician to watch over the subject of it, and to place him, if possible, under a duly regulated mode of life. Such a person must ever be looked upon and treated as an invalid, for it is impossible that any one so affected can follow out all the ordinary pursuits of active life, for any length of time at least, with impunity; and most assuredly a small accident is, in him, sufficient to wake up the inflammation once again, and once again to add fresh injury to the valves.

But the valvular disease is, as we have seen, the fruitful source of disorders in different organs and parts of the body; and when once the disease has

produced such a degree of disorganization in the valves as greatly to impair their function, these disorders become permanently fixed, and display themselves by the presence of a train of more or less severe symptoms. Consequently, the treatment of these disorders and the relief of the disturbed and oppressed organs now becomes the particular objects of our consideration. Medicine can here, happily, do much in giving temporary relief, and in diminishing the sufferings of the patient.

Pulmonary Disorders: Treatment.—Pulmonary affections are the most constant and the most important of the secondary disorders which result from valvular diseases. Their treatment is, necessarily, only palliative, inasmuch as the exciting cause of them (that is, the valvular disease) is in continual operation. The treatment has one immediate and special object in view, and that is to free the respiratory organs from the obstacles which, within themselves, oppose the due performance of their functions; and to allay as far as possible the disturbance of the function of the heart, which is their immediate cause.

The pulmonary affection varies much in degree in different individuals, and in the same individual at different times. It may at one time be represented by disturbances merely of a transient character,—by a passing congestion of the lungs; and at another time by the most formidable disorders. The injury

of the valves may, in fact, be such, that, under the ordinary conditions of a tranquil life, the subject of it is free from the usual symptoms which mark its existence; it being only under the influence of mental excitement, or of unwonted bodily exertion,—when the blood is suddenly forced in increased quantity towards the heart,—that the organ fails, and that congestion of the lungs is excited. In cases of this nature the treatment is simple and hopeful, even though the function of respiration be for a time much disturbed. When the heart's action is strong, and the bodily health vigorous, and the other organs of the body free from disorder, the mere cessation of the extra excitement often suffice to restore the lungs to their wonted condition.

But when the valvular disease has occasioned extensive structural changes in the lungs, the prognosis becomes much more serious, and we can no longer expect such beneficial results from treatment. The oppression of the lungs here is no longer the result of simple congestion. The disordered condition is of long date, and its cause—the valvular disease—as we must remember, is permanently in action, and under all conditions of the heart's action, whether tranquil or excited. Moreover, at this period of its progress, other disorders complicate the case,—abdominal congestions, serous effusions, and cerebral disturbances. The patient, through long suffering, and disturbance of the general functions of life, has

become weak, pallid, and anæmic, and the action of his heart is feeble, fluttering, and intermittent.

Congestions of the lungs, bronchitis, pneumonia, œdema, and pleuritic effusions—these are the pulmonary disorders which occur as consequences of organic valvular diseases. It is, therefore, impossible to lay down any particular rules which shall comprise the treatment of all cases of this description; for each individual case requires an especial consideration. We must remember, however, that what we are now aiming to obtain is temporary relief, not permanent cure, and that this relief must be sought at the least possible expenditure of vital power, and of the least shock to the constitution.

There are two objects which we have to pursue in carrying out the treatment of these pulmonary affections, viz.: to arrest the increase of the valvular lesion; and to remove the local impediment to the respiration which results therefrom, and which is represented by some one or more of the above-mentioned disorders. The first indication we fulfil by prophylactic treatment.

In carrying out the second, we attempt to tranquillise the disturbed and irregular action of the heart. By quieting its rapid, feeble, tumultuous, and irregular movements, the force and vigour of its contractions are considerably increased; and by thus tranquillising and regulating the action of the heart, we likewise diminish or arrest the cause which is pro-

ducing the pulmonary congestion. We also attempt the removal of the local impediments to the respiration which exist in the lungs themselves, viz.: congestions, serous effusions into the pleuræ and into the air-cells and tissue of the lungs, pneumonic and bronchitic exudations, and accumulations of mucus in the bronchial tubes.

The exudations of mucus and of blood from the mucous membrane of the bronchial tubes, which so often take place in these cases, are frequently sources of great relief to the congested lungs, and they may be encouraged so long as the patient has strength to eject them; but should his powers fail, he is thereby subjected to the danger of suffocation. It is better therefore not to trust too much to this natural effort of relief, and throw too great a task upon the lungs themselves. A few leeches from time to time, blisters, mustard poultices, stimulating liniments, should be applied to the thorax.

The excellent effects of small bleedings—the immediate and temporary relief which they occasion in this condition of cardiac and pulmonary congestions—are often very striking. I believe that a few ounces of blood, more or less according to the condition of the patient, taken opportunely and to this end, will very frequently prolong the patient's life, and save him from impending death. The bleeding, I believe, relieves and gives freedom of action to the distended heart and congested lungs. The object of the

bleeding in this case is very different from that which is supposed to indicate its use, generally, in inflammations. The modern prejudice, therefore, against bleeding in inflammation should not, as I believe it too often does, prevent the physician resorting to its use in cases of the nature which I am here speaking of.

The following case marks well the effects of this remedy:—

A powerful, vigorous-looking man, who had enjoyed good health previously to his present illness, came under my care in St. Mary's Hospital. He was suffering severely from dropsical symptoms; the lower part of his body was œdematous; and there was serous effusion in his peritoneum and pleura. His breathing was much oppressed and very painful. His face was dusky. The cause of all this mischief, which had arisen rapidly, was organic valvular disease of the heart. The remedies prescribed gave him no relief. On the third day of his residence in the hospital, he was evidently worse: he could not lie down. I ordered him at once to be bled, and stood by him while about twenty-six ounces of blood were taken from his arm. He felt immediate relief. The next day I found him decidedly better. On the third day after the bleeding, he was able to lie down in bed. In the course of a week, he was walking about the ward, and soon afterwards left the hospital convalescent, and, as he himself thought,

quite well, but, of course, with his organic disease of the heart unchanged.

In this case, the good effects of the bleeding were too immediate and lasting to leave any kind of doubt as to their reality; and I see not how it is possible to ascribe those effects to any other cause than to the influence exercised by the bleeding over the heart. There was here no inflammation to relieve; and it is very certain that the bleeding could have no direct power in removing the effusions which were mechanically pressing upon and impeding the function of the lungs and other organs. If it should be suggested that the bleeding promoted the action of other remedies, I would answer: that it doubtless did so, in that it relieved the heart, but that the benefits of the bleeding were distinctly manifested, before those remedies displayed their due effect.

Cases of this kind are to be carefully distinguished from those in which the pulmonary congestion has been gradually produced, by the pressure of serous exudations in the pleura upon the lungs, and by œdema of the lungs, &c.; for in such cases the disease is irremediable, and the pulmonary congestion the inevitable forerunner of death. We must remember, that there are congestions of the heart, and periods in the course of all congestions, in which no relief can be hoped for from venesection.

Œdema of the lungs partakes of the general dropsical condition of the body; it is a sign of weak-

ness, of advanced disease, and a bad sign. Œdema supervenes far on in the progress of the disease, and is an indicator of its fatal termination; and it is therefore little amenable to treatment—at least to any local treatment. Here we are placed between two dangers; if we attempt to remove the dropsical disorder, we are compelled to use remedies which weaken the general system, and therefore have a tendency to produce that very condition whose removal is attempted. In all these cases of advanced cardiac disease the use of stimulants properly regulated is absolutely necessary.

Abdominal Disorders: Treatment.—Numerous disorders of the abdominal organs arise as consequences of obstructed circulation through the heart. The liver, from its intimate connexion with the heart, especially resents the obstruction; and its engorgement, and consequent enlargement, are the result. When thus enlarged, it sometimes compresses the right lung upwards, and so interferes with the pulmonary functions; and in like manner, it may impede the action of the heart, by forcing the organ upwards and towards the left. The digestive and assimilative, as well as the secretive functions of the abdominal organs, suffer greatly from the continued congestions of blood to which they are subjected by these diseases of the heart. The congestions of these organs make themselves known to us, among other ways, by loss of appetite, and disorders of the diges-

tion and the nutrition. Hæmatemesis, hæmorrhage from the bowels, and diarrhœa, also not unfrequently occur, and occasionally give temporary relief. The congestion of the kidney diminishes the secretion of urine, and sometimes renders the urine albuminous. Thus, these secondary abdominal disorders in themselves tend to destroy life.*

Under these circumstances, we must pay attention to the function of the liver, and endeavour to maintain a free flow of the bile, remembering that the chief decarbonizing organs of the system—the lungs —are already seriously disordered, so that the proper performance of the function of the liver is here of especial consequence. For this purpose we must make use of salines and mercurials.

The gastric disorders arising from the congestion of the portal system are, unfortunately, difficult to treat; but their importance in deteriorating the powers of life, by preventing nutrition being properly performed, is evident enough. The congested state of the intestinal canal and of the kidneys, which is more or less their permanent condition in advanced stages of cardiac disease, impedes the proper assimilation of the food and the secretion of the urine (producing constipation and a diminished flow of urine),

* I need hardly remind the student, that all the symptoms here described result from the mechanical obstruction to the portal venous system, which is produced by the prevention of the return of blood to the heart by the cardiac disease.

and it also interferes with the application of the remedial measures which are requisite.

Besides these congestions, we have also to attempt the removal of the dropsical symptoms—the serous effusions—which are the results of the cardiac disease.

These effusions, we must remember, are accidents arising, for the most part, as attendants of heart diseases in their latter stages, and, therefore, when the system is already enfeebled by long pre-existing disease. Hence, in almost all cases, we must support the body, by stimulants and a nourishing diet, while endeavouring to maintain the flow of its different secretions. The milder warm cathartics, blue pill, colocynth, and aloes, in different combinations, and saline medicines, are the best adapted for the relief of the intestines. The use of elaterium, croton oil, etc., must be resorted to in those cases in which other remedies fail to give relief; and when used, their administration should be carefully watched. Bitartrate of potass with jalap is also an excellent remedy here. Acupuncture and incisions in the lower parts of the body are often of great temporary service; they give relief by draining off the serum in large quantities. Large intervals in the skin should be left between each puncture, to prevent inflammation.

In the anæmic condition of the body, produced by cardiac disease, and when dropsical effusions have taken place, I know of no remedy which, when it

can be taken, is so efficacious as a tonic and diuretic, as tincture of the sesquichloride of iron given in bitter infusion with tincture of digitalis.* Digitalis is also an invaluable remedy for tranquillising and regulating the action of the heart in hypertrophy resulting from advanced valvular diseases. I find no other of the preparations of steel equal in efficacy to the sesquichloride.

When the ventricles of the heart are hypertrophied and dilated, its impulse is usually increased in force, and altered in character. When the hypertrophy is considerable, instead of the ordinary beat felt at one point, we now both see and feel a forcible heaving forwards of the walls of the chest. The head of the auscultator is thus often perceptibly raised at each beat. The impulse, however, may be weak, and even imperceptible, notwithstanding the heart be hypertrophied, for rapidity and completeness of the heart's contractions are necessary to produce a strong impulse; and these conditions are not always

* Digitalis is dreaded because it is supposed to have a *paralysing* action over the heart. Dr. Handfield Jones has lately made some experiments on this point, and has shown me the heart of a cat poisoned by digitalis, spasmodically contracted, several hours after death. I may add, that I have used that remedy very largely indeed, and have never observed any evil consequences resulting from its use. I therefore regard the fear of using it, which still lingers in the minds of many practitioners, as quite chimerical. We must recollect that patients afflicted with cardiac diseases often die suddenly, whether they do or do not happen to have taken digitalis.

present, on account of the rhythmic movements of the heart being deranged by the cardiac disease.

An hypertrophied ventricle is not of itself sufficient to produce a strong impulse; in fact, when the ventricle is hypertrophied and contracted, the impulse of the heart is weak. The impulse of a simply hypertrophied heart does not produce the strong, slow, extensive heaving of hypertrophy and dilatation; it has more the character of a quick, sharp blow.

The impulse of the heart is stronger than natural when its walls are dilated without thinning, but it is weaker when the walls are at the same time abnormally attenuated; a ventricle thus dilated, has not sufficient force to expel its contents rapidly and completely.

When the right ventricle is hypertrophied and dilated, the impulse is often very great about the xiphoid cartilage, and at the lower portion of the sternum, and sometimes is communicated to, and strongly perceptible over, a considerable portion of the hypogastric region. When the left ventricle is hypertrophied, the impulse is felt more towards the left side of the thorax; but it must be recollected, that the cause which excites hypertrophy of the left ventricle, sooner or later produces, in a secondary manner, hypertrophy of the right ventricle also.

Hypertrophy and dilatation of the heart, associated with considerable contraction of the aortic orifice,

may give rise to a feeble impulse, on account of the heart's contractions not being complete. When the contraction is such as to admit of the complete expulsion of the blood from the ventricle, then the impulse is forcible and prolonged.

On the subject of the *treatment* of hypertrophy and dilatation of the heart, I need not linger. Hypertrophy and dilatation of the heart are almost invariably, if not invariably, secondary disorders, the consequence of valvular and other diseases; their treatment, therefore, necessarily becomes a part of the treatment of such diseases. I therefore refer the reader to the treatment of valvular diseases, and of the secondary disorders thence resulting.

Enlargement of the Thyroid Gland, with Hypertrophy of the Heart.—Before concluding this chapter, I may add a few words respecting a series of symptoms which, it has been said, have some especial connexion with an hypertrophied and dilated condition of the heart. I refer to the swollen state of the thyroid gland, the violent action of the cervical arteries, and enlargement and prominence of the eyes, occasionally found in association with permanent excitement of the heart. This affection has been particularly investigated by Dr. Graves and Dr. Stokes. The subject, however, is one which still requires further elucidation; the conclusions which have been arrived at concerning it being based upon a too slender series of observations.

The condition above stated, of irregular, rapid, and increased action of the heart, associated with throbbing of the cervical vessels, and enlargement of the thyroid glands and the eyeballs, is unattended with any sign of cardiac inflammation. It is generally seen in females, connected with hysteria and uterine disturbance, and occurs at any age above puberty. It is rarely observed in men. The symptoms have remissions, which appear to depend upon the state of the heart's action. The enlarged thyroid offers a throbbing pulsation, and the usual physical signs of aneurismal varix. These symptoms may be partial or general, varying in intensity at different parts of the tumour, and at different periods of the disease. With the progress of the disease, as the gland becomes more solid, they partially subside. Venous murmurs may be heard occasionally, over and around the tumour. The enlargement of the eyeball varies at different periods, and is not necessarily attended with any loss of vision. The essence of the disease, according to Dr. Stokes, consists in functional disturbance of the heart, which is followed by organic changes.

The few cases of this disease which have fallen under my own observation, lead me to think that Dr. Stokes' view of the nature of it is not correct. Neither do I think that his own cases sustain his opinion. I quite agree with Dr. Todd, that this is a special disease of which the cardiac disturbance is

merely an incident. Dr. Todd says of it:—"I have seen in so many instances, especially in women, the concurrence of prominent eyeballs, rapid and vehement action of the heart, and enlarged thyroid, that I have long looked upon this aggregate of symptoms as constituting a special disease." (Lect. 220.)

The following is a good example of the malady:—

Affection of the Heart, with enlargement of the Thyroid and Thymus Glands, and Prominence of the Eyes.

The subject of this disease was a female, æt. 26. Nine years before her admission into St. Mary's Hospital in March, 1858, she had suffered from rheumatic fever, and had ever since been subject to rheumatism. The thyroid gland began to enlarge six years ago. For the last six months she has been constantly under medical treatment for palpitations.

Her eyes were so prominent as to prevent her closing her eyelids, but she had never been troubled with conjunctivitis. The least exertion, mental or bodily, brought on violent palpitations, which were always attended with "dull, wearing pains." The beat of the heart was like that of an hysterical person. A slight systolic bruit was audible over the whole præcordial region. In the second left intercostal space, close to the sternum, a loud, rough, prolonged systolic bruit was heard and a thrill felt by the finger; *the murmur though loud was very limited,** and constantly present. The patient was highly nervous and excitable. The pupils were of

* I have another case of this curious affection under observation at the present moment, in which a similar systolic murmur exists, and at the same part.

equal size. From treatment she derived only temporary relief. Latterly her breathing became affected, and she died somewhat suddenly about two months after her admission into the Hospital.

P.M.—The body was very anæmic; the gums were quite blanched. The thyroid gland, large and firm, embraced, but did not compress, the trachea and larynx; the carotid arteries and internal jugular veins were much compressed by it. The thymus gland was remarkably enlarged, weighing two ounces and a half; it passed down along the anterior mediastinum, ending in two lappets, one of which, larger and broader than the other, *lay across the pulmonary artery, and apparently compressed it.* The structure of the gland was perfectly normal. The heart's valves were slightly thickened but competent. Its muscular structure was much altered; numerous little patches of firm white fibrinous matter were scattered through the walls of the left ventricle; the papillary muscles were particularly affected, being partially converted into a dense fibrinous structure. A few atheromatic spots were observed in the coronary arteries, and in the first part of the aortic arch. The lungs, posteriorly, were hepatized. The brain and its vessels were healthy; and the other organs of normal appearance.

CHAPTER XVI.

FATTY DISEASES OF THE HEART.

FATTY GROWTH ON THE HEART.—SYMPTOMS.—FATTY DEGENERATION OF THE HEART.—PATHOLOGY.—SYMPTOMS.—DIAGNOSIS.—PROGNOSIS.—TREATMENT.—RUPTURE OF THE HEART.—SYMPTOMS.—TREATMENT.

Fatty Growth on the Heart.—A certain amount of fat is found, in health, deposited upon the heart, and chiefly along the course of the coronary arteries and their branches.* Sometimes the fat accumulates to a morbid extent; that is, in such abundance, as by its presence to interfere mechanically with the free action of the organ. Such morbid accumulation of fat has received the name of *fatty growth* on the heart, to distinguish it from another morbid condition, namely, fatty degeneration of its muscular tissue. The fatty growth is more developed on the right than on the left side of the heart, and may be associated with a perfect, healthy state of its muscular structure. When, however, the growth of the fat is consider-

* For the history of fatty diseases of the heart, the reader must be especially referred to Dr. Quain's well-known paper in the 33rd vol. of the *Trans. Med. Chir. Soc.* His paper was the starting-point of modern investigators into this subject.

able, the muscular tissue is usually more or less altered, the fibres being softened and atrophied, and encroached upon by the fat around them.

There is no absolute connexion between general obesity and fatty growth on the heart; but the two conditions usually coexist, so that, when other symptoms lead us to expect the presence of fatty growth on the heart, the existence of general obesity strengthens the diagnosis.

Accumulation of fat on the heart takes place under two opposite conditions of the body; for there is what may be called a *sthenic* and an *asthenic* form of obesity. *Sthenic* obesity is a simple hypertrophy of the fatty structures of the body, occurring in individuals in the prime of life, and who are otherwise healthy. Here the fat accumulates on the external parts of the body, as well as around the internal organs; just as it does in the instance of animals fattened for the market. In *asthenic* obesity, on the other hand, the fat tends rather to accumulate upon the internal organs. This condition, moreover, occurs chiefly in persons of advanced life, or of broken-down constitutions, and is connected with decay of the vegetative life, with depravation of the blood, and with weakness of the assimilating processes. In the sthenic form, the fat is accumulated chiefly about the base of the heart; in the asthenic, it collects towards the apex, and is deposited at the expense of the muscular tissue, rather than accumulated upon it.

Sthenic obesity appears to exert a predisposing influence to fatty degeneration, but may long exist unassociated with any structural change. Asthenic obesity is, on the other hand, closely associated with fatty degeneration; and where the one condition exists, we may suspect the coexistence of the other likewise. (Dr. H. Jones.)

Symptoms.—A certain amount of fat may be deposited upon the heart's surface without interfering with its functions. But when large collections of fat take place upon it, or dip down between the muscular fibres, an atrophous condition of the tissue will at last result; and hence fatty degeneration is thus frequently associated with fatty growth on the heart. We do not possess a clinical history of the symptoms of simple fatty growth on the heart. The cases described by the older writers are valueless, inasmuch as we have no proof that in them the muscular fibres were intact and sane. The symptoms present in one case, well observed by Dr. Walshe, were: sensation of oppression at the præcordium, difficulty in walking quickly, cold extremities, feeble pulse, giddiness, weak cardiac impulse, extensive dulness on percussion over the heart, whose sounds were weak. The symptoms were in fact those of a heart impeded in the performance of its functions.*

* Corvisart refers to the case of a child, related by Kerckring, in which the heart was so enveloped in fat as to appear altogether absent. The child was extremely fat, and died suddenly

Fatty Degeneration of the Heart.—This form of disease differs entirely from fatty growth on the heart. Here the intrinsic change consists, not in a mere deposit of fat on or between the fibres of the heart, but in a morbid condition of the muscular fibre itself—its sarcous elements being converted, wholly or partially, into oily matters. When the muscular fibre, thus diseased, is viewed under the microscope, we find that its sharp, well-defined outlines are lost, that its striæ have disappeared, and that oily granules occupy the place of the striæ and their intervals. In cases of far advanced degeneration, oily particles may also be seen scattered between the muscular fibres in the field of the microscope. These changes take place gradually, so that the disease may be met with in different stages of progress at different parts of the heart. When the degeneration is far advanced, whole fibres appear fused together, losing all trace of resemblance to the natural tissue. It may be partial, affecting only certain portions of the heart; or the degeneration may affect some of the fibres in all parts of the organ. It attacks the tissue of the left more frequently than that of the right ventricle of the heart; but usually both ventricles are affected

from suffocation. When the accumulation of fat on the heart is very great, it mechanically impedes the action of the organ. We see this in the case of prize-fed animals, in whom extreme breathlessness is induced by the least exertion—a condition which occurs before the muscular fibre becomes degenerated.

simultaneously. The auricles appear to be less frequently injured by the degeneration, than the ventricles.

When the degeneration is general, and has reached a somewhat advanced stage, the naked eye detects alterations in the physical appearances of the heart. Instead of the bright red colour of health, the muscular tissue presents a pale, fawn, yellow, dirty, mottled aspect. The firmness of its structure is lost; the organ becomes soft and friable, so that it may be torn or ruptured with great facility. Its ventricular walls have a loose, flabby, doughy, and inelastic feeling; and may sometimes be doubled-up, and moulded by the slightest pressure; falling together when cut across. It is almost always in connexion with this softened and lacerable condition of its tissues, that rupture of the heart occurs.

Fatty degeneration of the heart is met with in persons of all ages, and of both sexes, but more often in men than in women. It occurs most frequently at advanced periods of life, and then, at times, apparently, as one of the ordinary consequences of old age. All classes of society are subject to it; the luxuriously fed and the ill-nourished appear equally obnoxious to its slow and insidious attacks.

Sometimes the fatty degeneration of the heart constitutes the only perceptible diseased condition existing in the body after death, but more generally it is associated with other structural changes: with fatty

diseases, for instance, of other organs and parts; with diseases of the kidney; with atheroma of the arterial trunks and their valves; with hypertrophy —but more frequently dilatation—of the heart; with pericardial adhesions and other pathological indications of previous attacks of pericarditis and endocarditis; with apoplexy, with emphysema of the lungs, &c. &c.

Fatty degeneration appears, however, to be in some way closely associated with calcification of the coronary arteries, the two diseased conditions being very frequently found existing together. It has even been observed, that where the heart's tissue was healthy at one part, and degenerated at another, that the part degenerated was connected with a branch of the diseased coronary artery.

In cases of this kind, it appears evident that the muscular tissue degenerates, because its arterial supply is defective. And it is worthy of remark, that exactly the same series of pathological changes, defective nutrition, and softening appears to take place here as occur in softening of the brain in connexion with a calcareous and fatty condition of the cerebral vessels. So that, indeed, there exists a remarkable analogy between the degenerations, which are usually associated with apoplexy of the brain and with rupture of the heart.*

* Mr. Swan (*Lond. Med. Gaz.* Nov. 1848) has observed: that "although the coronary arteries communicate at the base and

But fatty degeneration exists independently of this calcified state of the coronary arteries; and in such cases we must regard it as the expression of an enfeebled and unhealthy condition of the body—in fact, as of constitutional origin. This view is confirmed by the frequency with which diseased states of other organs are observed, in connexion with fatty degeneration of the heart.

Symptoms of Fatty Degeneration.—We have seen that the pathological condition which marks the existence of this disease of the heart, consists in the degeneration of its muscular fibres, and in proportion to the extent of the degeneration which its tissue has undergone. Thereby the physiological action of the fibre is diminished in force or destroyed, and the contractile power of the heart, consequently, more or less enfeebled. Accordingly, we find that the chief symptoms attending this condition of the heart are those which indicate defective condition of its contractile power.

These symptoms are: general debility; incapacity for exertion; shortness of breath, sometimes slight, sometimes very severe; weakness of the digestive

apex of the heart, the communication is not very free, and each can do very little more than supply its respective regions." So that if a coronary branch is obstructed, the part to which it is distributed will not receive an adequate supply by anastomosis from the other branches. A similar state of things holds good in the case of the cerebral arteries. Beyond the circle of the Willis the arteries have very little communication.

powers; stifling sensations, with flutterings, occasionally felt over the heart, accompanied with more or less of pain. The impulse of the heart is weak; its sounds are feeble. Cardiac bruits also may be accidentally present, but they have no necessary connexion with fatty degeneration. Severe cardiac pains, united with shortness of breath and syncope,—the combination of symptoms which represent angina pectoris,—are also observed in advanced conditions of this disease. (See Angina Pectoris.) They indicate a sudden interference with—partial interruption of—the action of the heart, such as may produce a fatal termination. The cerebral disturbances attending fatty degeneration of the heart, vary between a passing giddiness and complete coma; they are, for the most part, but slight and transient, and quickly pass away. They may be attributed to the weakened action of the heart, which, under slight disturbing influences, becomes incapable of properly carrying on the cerebral circulation. Sometimes the attacks are severe, and then the patient may fall as if struck by apoplexy; but the weak pulse, the pale features, the unaltered pupils, the absence of stertor, the advanced age of the patient, serve to distinguish the attack from true apoplectic seizure. The attack, moreover, is rarely followed by paralytic symptoms.

The existence of the "*arcus senilis*"—a fatty degeneration of the outer border of the cornea—was first pointed out by Mr. Canton as a sign corrobora-

tive of the existence of fatty degeneration of the heart. There can be no doubt, however, that its value as a sign has been much overstated. The frequency with which the arcus senilis is met, in the most healthy individuals in advanced life, justifies us in considering it in them as little more than a natural consequence of old age. From a general review of the facts known on this subject, I am led to conclude that arcus senilis is, in old people, in the absence of other symptoms, a sign of little value as an indicator of fatty degeneration of the heart; but that when present in a marked degree, associated with other symptoms, it corroborates the diagnosis of the fatty degeneration: and that in early life it is much more frequently absent than present in cases of fatty degeneration of the heart.*

Diagnosis.—There are no signs which positively demonstrate the presence of fatty degeneration of the heart; but in its advanced stages, and when it occurs at a late period of life, the rational signs of the disease are, for the most part, tolerably well marked. In early life, however, and when the degeneration is slight, the diagnosis must always be very conjectural, and the disease, consequently, may then be readily overlooked. Besides this, it must not be forgotten, that fatty degeneration of the heart, in an advanced stage even of its progress, has been found in persons

* I have frequently in the deadhouse noticed the coincidence of *arcus senilis* with a perfectly healthy condition of the heart.

who have died suddenly, and in whom, during life, no symptoms had existed such as to excite suspicion of the presence of any cardiac disease in the individuals themselves. Possibly, however, some morbid indications might have been discovered in these cases had they been brought under the notice of medical observers.

Diagnosis of this disease is mainly based upon a consideration of the symptoms which result from, and are indicative of, an enfeebled heart—enfeebled through diminution of its muscular power, and so rendered incapable of duly regulating the movements of the blood, and supplying the wants of the system.

The *prognosis* of the disease must be always unfavourable. The degeneration, as we have seen, may sometimes remain latent almost up to the moment that it produces death; and whenever it has arrived at such an advanced stage, as, by symptoms, to indicate its existence, we may be sure that the destruction of the muscular tissue is great; and we know that its repair is impossible.

Treatment.—Whenever the symptoms in any case are such as to lead us to suspect the existence of fatty degeneration of the heart, we have three main objects before us for consideration in its treatment: viz., to prevent the further progress of the degeneration of the tissue (its repair being, reasonably, supposed, impossible), by removing all causes which tend to exhaust and weaken the bodily or mental

powers, and by improving the nutritive functions; to regulate the habits of the patient, so as to protect him, as far as may be, against the dangerous consequences, to which, under such a condition of the heart, he must ever be liable; and, thirdly, to alleviate and remove, if so it may be, the angina-like attacks which are associated with advanced conditions of the disease, and are, indeed, sometimes its fatal conclusion.

A generous and stimulating diet, pure air, moderate exercise when it can be taken without inconvenience, and due regulation of the digestive organs, with tonic medicines, particularly steel in bitter infusions—in fact, the use of all those means which tend to strengthen the constitution—are to be recommended, for the first object. The use of oils has been proscribed; but on the insufficient grounds of their fatty nature. I have certainly seen cod-liver oil, combined with steel, followed by very satisfactory results in supposed cases of fatty degeneration of the heart.

The paroxysmal pains, the angina-like attacks, demand antispasmodics and stimuli; when very severe, small abstractions of blood and blisters may assist in relieving them. Cerebral and syncopal symptoms—giddiness, fainting, vertigo, &c., must be treated by internal stimuli, and by the application of counter-irritants to the surface of the body.

Violent bodily exertion and mental emotions should be carefully avoided: all efforts, in short, which call

ıpon the heart for sudden and unwonted energy of 'unction.* The heart, being inordinately weak, may ɔossess power enough to carry on the circulation]uietly, but a slight extra strain throws the organ sud-lenly off its balance, and so as to endanger and even lestroy life. "The patient," as Mr. Paget says Lect. i. 129), "may be fit for all the ordinary events ɔf calm and tranquil life, but is unable to resist the torm of sickness, or accident, or an operation." In ases of this kind, where we suspect the existence of 'atty heart, we should be extremely cautious in the dministration of chloroform.

Rupture of the Heart, when not caused by external njury, is chiefly known to us in connexion with fatty legeneration of its muscular tissue. The accident nay, indeed, result from other causes, such as from n abscess in, or aneurism of, or hæmorrhagic effusion nto the walls of, the heart; or from their ulceration, nd dilatation with attenuation, but such causes are xceedingly rare. Under all circumstances, how-ver, we may be sure that whenever, so-called, spon-aneous rupture of the heart occurs, the muscular

* The effects of exertion after food are well shown in the fol-ɔwing case of death from fatty degeneration of the heart (*Path. Trans.* vol. vi.) :—"Sir J. M., æt. 48, arrived in town in appa-ently excellent health, having only a slight cough. Next morn-ıg, immediately after a hearty breakfast, he went out, and after few minutes fell insensible in the street. He was seen, within wo minutes, by Mr. Moore of the Middlesex Hospital, but was lready dead."

tissue is not in a healthy condition. It is impossible to imagine that rupture, such as occasionally occurs to the abdominal muscles in tetanus, can happen to the muscular structure of the healthy heart, as a consequence of the violence of its contractions.

Rupture of the heart occurs more frequently in the male than in the female sex, and almost invariably at advanced periods of life. All parts of the heart are subject to laceration: the ventricles, the auricles, the septum, the papillary muscles and their tendons; but of all parts, the ventricles are most liable to the accident, and the left ventricle much more so than the right. The rupture is in most cases single, but occasionally it is represented by several small lacerations; the passage it forms is generally direct, but now and then it runs obliquely and sinuously through the walls of the cavity, and it has been known to extend from the apex to the base of the ventricle. The rupture may occur gradually, the blood insinuating itself between, and progressively forcing its way through, the softened muscular fibres. Usually, however, the rupture is completed at once. The most frequent seat of the laceration is about the middle of the anterior part of the left ventricle, near the septum. The rupture, most probably, occurs at the time when the muscles are acting most vigorously, *i.e.* at the commencement of the systole. Ruptures of the valves and their tendons are in most cases the results of endocardial inflammation; and have, there-

fore, a different signification from rupture of the heart, which occurs as the result of its fatty degeneration. (See Appendix V.)

The immediate cause of the rupture may, in most cases, though not in all, be traced to some inordinate or sudden strain upon the heart's action, produced by bodily exertion or mental emotion, such as in its enfeebled deteriorated state it is unable to sustain. But the original cause is to be sought in the diseased condition of its muscular structure.*

Death is generally, but not always, the immediate result of the rupture. Corvisart refers to a case of ruptured heart caused by external injury, where the patient survived, until the twenty-third day, the infliction of the injury. The sufferer may linger on for many hours; and, in most cases, appears to die at last not from loss of blood (for this is rarely very great, on account of the indistensible structure of the pericardium), but from the pressure exerted upon the heart, and the arterial trunks arising from it, by the blood which has escaped into the sac. By this

* In cases of rupture of the heart it is well to recollect, that there very frequently coexists an atheromatous condition of the coronary arteries; and where this exists, we may conclude, that the arteries generally, and especially the smaller vessels at the periphery of the body, are also diseased. Consequently, there is an obstruction to the circulation thus established; and the heart, already weakened by fatty degeneration, has therefore extra work to perform, at a time when it is least capable of performing it.

pressure the action of the heart is so interfered with, that it becomes at length incapable of carrying on the circulation. When death does not follow immediately upon the occurrence of the injury, the escape of blood from the heart takes place gradually and at intervals; and in such case the rent in the heart's walls is probably oblique, or it may be partially or temporarily obstructed by a coagulum.*

Cases like that given by Corvisart, in which firm coagula have been found in the opening, have suggested the possibility of the rent being permanently healed under favourable circumstances. The possibility of a cure has likewise been suggested by the fact that the marks of wounds, and even bullets, have been found in the hearts of individuals, and of the lower animals, who have died long afterwards from other diseases.†

It must be remembered, however, that the cases of spontaneous and of traumatic rupture, differ in this important particular, viz., that in the latter the injury occurs in a healthy, and that in the former it

* Rostan, also, found by the side of a newly-formed rupture, traces of an older one, which was firmly closed by a fibrinous clot. (*Mém. sur les Ruptures du Cœur. Journal de Méd., Juillet* 1820.)

† Boyer (*Mal. Chir.* 5, 605) relates the case of a soldier who was shot in the chest. He recovered, and died six years afterwards from some other disease. The ball was found imbedded in the apex of the right ventricle. In cases of this nature, however, the wound does not appear to have completely traversed the walls of the heart.

occurs in a highly diseased structure, and therefore in a structure very ill adapted for undergoing the process of repair.

Symptoms.—The symptoms attending this accident scarcely ever enable the physician to predict with certainty the nature of the injury which has occurred; for they are such as may result from other diseases of the heart and its great vessels. They indicate direct, sudden, and serious interruption to the heart's action. When death is not instantaneous, the patient complains of violent, oppressive, and stifling sensations at the præcordia—of pains shooting through the chest; he is very pale; his skin is cold, and covered with clammy sweat; the pulse weak and fluttering, and irregular; the countenance expressive of intense anxiety and suffering; and faintings, syncope, and convulsions sometimes supervene, as forerunners of death. Little faith can be attached to increased præcordial dulness on percussion, as a means of diagnosis, in cases of rupture of the heart, where death takes place rapidly. The dull percussion-sound, however, and absence of the heart's impulse, may assist the diagnosis, when death occurs at an interval of some hours after the accident.

In cases of rupture of the valves, of the tendinous chords, or of the papillary muscles, we observe the sudden appearance of severe symptoms of heart disease, together with, usually, a very loud endocardial murmur.

Treatment.—For the remedy of such an injury as rupture of the heart, medical aid has little to offer. Indeed, as we have observed, while life lasts, we can never be certain of the real nature of the injury which has produced the symptoms. The urgency and character of these symptoms can alone guide our treatment. To sustain, by stimuli, the system under the violence of the shock which it has undergone; to relieve the obstructed circulation of blood through the heart, and at the same time to tranquillize its action if excited—these are the indications which the physician must endeavour to fulfil, when opportunity is given him. In the majority of cases, the effusion of blood is rapid, and its pressure upon the heart so great as at once to destroy life. Hence it is only in those cases where the effusion takes place very gradually, or intermittingly, that we can entertain any small hopes of success.

CHAPTER XVII.

ANGINA PECTORIS.

PATHOLOGY.—CONNEXION WITH FATTY DEGENERATION OF THE HEART, AND DISEASE OF THE CORONARY ARTERIES.—RESEMBLANCE BETWEEN THE SYMPTOMS OF THE DISEASES.—ITS NATURE.—TREATMENT.

Much obscurity once hung over the pathological history of that peculiar and striking combination of symptoms, to which the name of angina pectoris has been given. The symptoms of the disease clearly indicated to earlier observers, that the heart was the seat of the affection; but yet no abnormal conditions of the organ could be discovered to which it could be especially traced. The angina was found to accompany all the known diseases of the heart, and it was found to be present in cases in which, after death, the organ to all appearance seemed perfectly healthy.

This obscurity has, however, been removed by more recent researches. We are now, by the aid of the modern microscope, able to recognise alterations in the elementary structures of parts which were necessarily unknown to earlier pathologists. And

we are thus surely led to the conclusion, that organic disease of the heart is never absent where the symptoms of angina pectoris are present. We find, moreover, that there is one particular condition of the heart, viz., its fatty degeneration, to which, in the absence of valvular and other diseases of the heart, the angina may very reasonably be ascribed; and this condition requires the microscope for its demonstration.

I have said, that angina pectoris had been noticed by observers to exist in association with all the recognised abnormal states of the heart; and if we look into the history of the subject, we shall observe that it has been at various times ascribed by different writers to each one of those states. There was, however, one particular abnormal state of the heart—namely, calcification of its coronary arteries—which was found more frequently than any other to exist in connexion with the symptoms of angina; and this diseased condition of the arteries was, consequently, considered by the majority of observers to be the cause of the angina. Now, it happens, as we have seen, that a very close relation is found to exist between calcification (or partial obstruction of the coronary arteries) and fatty degeneration of the muscular tissue of the heart, the latter condition being very frequently associated with the former, and apparently in some degree dependent upon it. The fatty degeneration has indeed been sometimes ob-

served solely in that particular portion of the muscular tissue, to which a calcified branch of the coronary artery was found to be distributed.

Symptoms.—The connexion of angina pectoris with this fatty condition of the heart, thus indicated by pathological anatomy, is likewise confirmed by the symptoms of the disorder. The paroxysms which occur in well-marked cases of fatty degeneration of the heart, resemble those of angina pectoris. Sudden, violent, rending, oppressive, and even agonizing pain at the epigastrium, extending more or less over the thorax, the pain passing down the arm and reaching even to the fingers; a weak and almost imperceptible pulse; hurried respiration and shortness of breath, approaching to choking and suffocation; pallor of the face, which is bedewed with perspiration, and expressive of extreme suffering, consciousness the while often remaining undisturbed; or occasionally syncope and coma, and even sudden death itself—these are all symptoms alike of fatty degeneration of the heart in its exquisite form, and of angina pectoris.

And there are other points of resemblance between them. Both disorders are rare, comparatively speaking, in females; both occur chiefly in men, and at advanced periods of life; both may be associated with every kind of organic disease of the heart. An attack, in both instances, is generally induced by excitement of the mind, or some unusual exertion of

the body—that is to say, by causes which suddenly disturb and increase the heart's action; and for this reason, also, it occurs more commonly in the day than during the night. In both, during the intervals of the attacks, the patient seems tolerably free from disorder. In both, one attack is pretty surely, sooner or later, followed by another, and on each occasion it recurs at shorter intervals and with an increase of severity. Both have, sooner or later, but one issue, and that a fatal one. The treatment required in both is alike. Antispasmodics and diffusive stimuli during the attack; quietude of mind and body, nourishing diet, and careful regulation of the animal functions during the intervals. These facts, I think, inevitably lead us to the conclusion: that there is a close connexion between the symptoms of the disease called angina and of fatty degeneration of the muscular structure of the heart.

The characters of an attack of angina pectoris, its sudden onset, and the apparently total disappearance of the disease when the attack has passed over, have induced observers to consider it as spasmodic—in fact, to ascribe the symptoms of the angina to a spasm of the heart. This view of its nature is, however, purely hypothetical, and certainly it is difficult, *à priori*, to understand how a spasmodic contraction of the muscles of the heart can be compatible with existence. The whole history of this disease quite opposes the idea of the angina being simply a spasm

of the heart; it tends rather to show the existence of a very opposite condition of the organ. The angina occurs mostly in the weak, and in those advanced in life. The heart, moreover, after death, is almost invariably found softened, flabby, fatty, attenuated, and dilated, and never spasmodically contracted, such as it has been seen, for instance, in tetanus; and such as we might expect to find it, if angina were indeed a spasmodic affection of the organ.

In corroboration of the views here given of the nature of angina pectoris, I may observe, that we not unfrequently meet with sudden cardiac attacks, closely resembling those of angina, which arise in the progress of recognised organic diseases of the heart, of contracted mitral orifice, or of deficiency of the aortic valves, for instance; the subject of them, during the intervals of such attacks, remaining in a tolerably good condition of health.*

For the ordinary purposes of tranquil life, the power of the heart, thus weakened by disease, may suffice to carry on the circulation; but when an extra stress is laid upon it—when, either through bodily exertion or mental excitement, an unwonted

* In the 8th vol. of the *Path. Trans.* I have recorded a case of aortic valvular disease and hypertrophy of the left ventricle, in which there had frequently occurred marked attacks of angina. In this case, the muscular structure of the heart and its coronary vessels were perfectly healthy.

quantity of blood is forced towards the heart—its powers prove unequal to the effort required of it. The blood accumulates in its cavities, the circulation is more or less arrested, respiration is necessarily affected, and then the train of painful suffocative symptoms—called angina pectoris—ensue; and even death itself may follow, if at last the heart be unable to struggle out of the violence of the attack.

And the fact is, that we observe symptoms, approaching more or less closely to those of angina, in most heart diseases, whatever their kind, when the disease has arrived at the stage in which the heart's action begins to fail, the blood to accumulate in its cavities, and the respiration to become suffocative—that is, when the death struggle has commenced. There are here the same rending pain, the same feeling of constriction over the heart, the same choking respiration, and then the same syncope and coma. The difference between the two cases appears to lie chiefly in this—that in fatty degeneration the angina arises suddenly, and suddenly passes away; whilst in these other heart diseases, it is the continuation of a struggle, and the violent termination of it. But there is in this nothing to contradict the idea, that the actual state of the heart, which produces the symptoms, may be alike in both, *so long as the struggle lasts.* In the one case, the heart for a season regains its former state, the paroxysm passes away, and the patient recovers; but he recovers, be it observed,

only to be again and again the victim of his malady —for one attack is invariably the forerunner of another, and each attack more severe and more readily excited than the former one. In the other case, the paroxysm is the last effort of the enfeebled heart to retain life. In the one case, the struggle occurs at intervals; in the other, it is a continuous struggle.

Corroborative of this it may also be observed, that an attack of pure angina pectoris, attended with all its characteristic signs, is a thing which rarely meets the physician's eye at the present day. Heberden speaks of it in his time as being "not extremely rare." The number of recorded cases, indeed, of so-called angina pectoris is much fewer now, since science has advanced the pathology of heart diseases, than it was some few years ago. "Many cases," as Dr. Stokes observes, "denominated angina pectoris by one physician, would be called cardiac asthma by another."

Angina pectoris, therefore, may be associated with, and be the result of, weakness of the heart, produced by an actual degeneration and destruction of the muscular fibres of the heart. Or, again, it may be present, when the muscular tissue remains healthy, but has lost its proper contractile power, through various causes, as above explained—in both cases the resulting cardiac disturbances, and the symptoms representing them, being alike.

Treatment.—The nature of this affection clearly

indicates what its treatment should be. In most instances, the violence of the attack has passed away before the physician can interfere. The powerful and sudden overwhelming of the vital powers is such as evidently to demand the immediate administration of diffusive stimuli—among which, warm brandy-and-water, the compound spirits of sulphuric æther, and aromatic spirits of ammonia, rank first in utility and efficacy; the object being to excite the contractions of the heart. If the pain be great and lasting, opium in full doses, repeated at short intervals, until relief is given, should be administered; for pain itself, prolonged and agonizing, will destroy life. But the most important part of the treatment is prophylactic. The great object must be to ward off the recurrence of the attacks; and we can, indeed, do much towards effecting that desirable end.

When the attack has passed away, the condition of the organs, and the general state of health of the individual affected, should be taken into consideration, and, as far as possible, every promoting cause of disease removed. All sources of mental excitement, and violent bodily exertion, should be most especially avoided, because these are found by experience to be the chief provoking agents of an attack.

The diet should be that which supports and invigorates the system generally, and tends to counteract or arrest that ill-nutrition which is supposed to

be the cause of the disintegration of the tissues of the heart. The disease is atonic, not sthenic.

To sum up the treatment in a few words, I may say that, during the attack, the heart's embarrassed action must be relieved, and its power sustained, by the administration of the most energetic and rapidly-acting stimuli; and in their intervals, any accidental disorder cared for, and the general health preserved, by strict attention to proper hygienic rules; all promoting and exciting causes of disease being most carefully avoided. If the presence of organic disease of the heart be discovered by physical signs, coincidentally with the angina, then that particular treatment will be required which would be considered proper in such disease.

CHAPTER XVIII.

CYANOSIS.

NATURE.—PATHOLOGICAL STATES OF THE HEART ACCOMPANYING IT.—CHIEFLY A CONGENITAL AFFECTION.—SYMPTOMS.—EFFECTS ON DIFFERENT ORGANS.—PHYSICAL SIGNS.—TREATMENT.

THE term Cyanosis, strictly speaking, implies a blue discoloration of the skin; and in such sense, all diseases which prevent the return of the blood from the systemic blood-vessels, and produce discoloration of the skin, through venous congestion, may be classed under this head. The word, however, is used in a more restricted sense, and serves to indicate the existence of certain deficiencies—for the most part congenital—in the anatomical construction of the heart, or of its great vessels, in connexion with which blueness of the skin appears as a constant and striking phenomenon.

Different accounts have been given of the cause of the blue discoloration of the skin. It has been ascribed to the admixture of arterial and venous blood, resulting from the existence of an abnormal

communication between the two sides of the heart, or between its great vessels. More extended observation, however, tends rather to show that the discoloration does not depend so much upon this admixture of blood, as upon the venous congestion which is associated with it, and which is the necessary result of the coexisting malformation or disease of the heart and its great vessels, or of the lungs. This conclusion appears unanswerably supported by the following facts, viz. :—That cases of defective organization of the heart, permitting of free mixture of the arterial and venous blood, are constantly met with, in which cyanosis is not present, provided the pulmonary artery is free and patent; That whenever cyanosis exists in a marked degree, constriction of the pulmonary artery is almost invariably associated with the defect of the heart; That contraction of the pulmonary artery, or of its orifice, must act powerfully and directly, as a cause of systemic venous congestion; And that cases of marked cyanosis have been observed, in which, after death, no communication was found to exist between the two sides of the heart, and in which, therefore, no admixture of blood could have taken place during life.

Thus it appears, on the one hand, that complete admixture of the arterial and venous blood may occur—as in the case where the heart possesses only a single ventricle, or where the descending aorta is given off by the pulmonary artery—without pro-

ducing cyanosis, provided the pulmonary artery be free; and again, on the other hand, that marked cyanosis may attend a constricted pulmonary artery, when no communication whatever exists between the two sides of the heart.

Hence, we may conclude, that the chief promoting cause of cyanosis is obstruction to the systemic venous circulation, produced by contractions of some one or more of the heart's orifices, and, in particular, by contraction of the pulmonary artery or its orifice. Free mingling of the venous and arterial blood within the heart may promote the cyanosis, but the intensity of the cyanosis and of the accompanying symptoms, viz. the difficult respiration and labouring heart, seem to bear a distinct relation, not to the extent of communication which may exist between the two sides of the heart, but to the degree of impediment which the above-mentioned constrictions oppose to the circulation.

Nor does there appear to be anything in the general history of the disease which contradicts this view. It has been asked, as an objection to the opinion that cyanosis is commonly the result merely of venous obstruction, how it happens that "the most intense venous obstruction may arise in the adult, without inducing cyanotic congestion?" The question may be answered by saying that the two cases do not admit of comparison. The intense venous obstruction here spoken of, is, in most cases,

the result of rapidly-induced disease; while the cyanosis, being of ancient date, the quantity of the blood, and the demand for its supply, have gradually accommodated themselves to the defective condition of the body, which is always associated with cyanosis. Moreover, in cyanosis, it is probable that the extreme vessels of the surface have become gradually enlarged, so as to admit of ready and rapid distension, to a degree which can be hardly supposed to exist in an ordinary case of venous obstruction.

Dr. Peacock, in his valuable treatise on "Malformations of the Heart," for the most part adopts these conclusions. He says (p. 126): "From these considerations, we are, I think, warranted in inferring that the cyanosis is due to congestion of the venous system." He considers, however, that the lividity, in cases of malformation, differs from that which attends ordinary disease of the heart or lungs. "That in cases of pulmonary and ordinary cardiac disease, the cyanosis is generally so much less intense than where the heart is malformed, is probably to be ascribed to the amount of congestion being less. In cases of acquired disease, were so small a proportion of blood submitted to the influence of the air, and were the general congestion so extreme, as in many instances of malformation, life could not be maintained. In acute affections, also, the integuments generally become more or less œdematous, so that the lividity is marked."

The inference deduced from his facts, by Dr. Peacock, is this: that the essential cause of cyanosis is obstruction to the flow of blood through the lungs, or from or into the right ventricle, but that the intensity of the lividity and its peculiar colour are modified by other circumstances. These circumstances are: the congenital existence of unnaturally dilated capillaries; the condition of the integuments, the body being generally emaciated, and the skin thin, when the peculiar colour of the dark blue disease is well marked, and an opposite condition of the body being observed, when the discoloration is of a deep rose tint; and lastly, the colour of the blood in the vessels, which must be necessarily dark in those cases in which only a small part of it is submitted to the aërating influence in the lungs. (P. 128.)

The most ordinary pathological condition of the heart met with in association with cyanosis is, the permanent opening of the foramen ovale, and of the ductus arteriosus—the openings being such as to permit of the free passage of blood between the two auricles in the one case, and between the pulmonary artery and the aorta in the other. At the same time we must remember, that in both these parts the opening may remain unclosed to a certain extent, without giving rise to any abnormal symptom whatever, provided the orifices of the heart and the trunks of the great vessels be not contracted. Patency of

he ductus arteriosus seems always to coexist either vith an open foramen ovale, or with perforation of he ventricular septum. Besides these, we meet with he following conditions in connexion with cyanosis: —Deficiency of the intra-ventricular septum; Mal-position of the origin of the aorta; The origin of he pulmonary artery and aorta from one ventricle, he heart being in such case formed of one auricle nd one ventricle only; Transpositions of the great vessels,—the aorta arising from the right, and the pulmonary artery from the left ventricle; The origin of the aorta and pulmonary artery from both ventricles simultaneously, the semi-lunar valves being in such case defective and ill-formed.

Together with these abnormal conditions of parts, there almost invariably coexists an obstruction of some one or more of the orifices of the heart, or of its great vessels, that of the pulmonary artery being the one most frequently met with. These contractions seem to determine, as we have seen, the existence of the cyanosis, and the admixture of the arterial and venous blood through the abnormal opening: as, for instance, through the foramen ovale and ductus arteriosus; and, of course, they do so by obstructing the current of blood. When these parts remain open to a certain degree, and no constriction of the orifices exists, the admixture does not appear to take place.

Cyanosis is for the most part, but not entirely, a

congenital affection. Dr. Peacock (p. 118) finds, from an analysis of 101 cases of malformation of the heart, that in 74 the symptoms were observed either at the birth, or immediately after; that in 15 they appeared before the end of the first year; in 1 at the sixteenth month; in 3 at two years; in 2 at three years; in 1 at three-and-a-half years; in 2 at five years; in 1 at eight years; in 1 at thirteen years; and in 1 at fourteen years, of age. It has occasionally been known to appear at a late period of life; and in such case its presence has been explained by the supposition that some violent action of the heart has suddenly enlarged an already existing patency of the foramen ovale; or that an intra-ventricular or intra-auricular communication has resulted from the rupture or the ulceration of the septum between the auricles or the ventricles. It has been known also to follow as the consequence of a violent blow over the heart.

Symptoms.—The discoloration, dependent, as we have seen, on venous congestion, necessarily varies much, both in its intensity and in the extent to which it affects the surface of the body. It may be limited to certain parts, and it may be general; but it is always most striking in those parts which present naturally a ruddy hue, as the cheeks, the lips, the nails, the inside of the lips, etc. It varies much also in the same individual at different times, becoming much more marked than ordinary when the heart's action

is excited by muscular exertion, or mental emotions, or when any intercurrent pulmonary affection produces impediments to the respiratory function. The temperature of the surface of the body is generally lower than natural, and the physical and moral energy of the patient defective. He is also very susceptible to alterations of temperature, and suffers much from cold. The extremities of the fingers usually take the clubbed form, with incurved nails, so commonly observed in phthisical subjects.

These symptoms naturally vary much in degree in different individuals, according to the different kind of abnormal changes from which they result, and the extent which the abnormal changes have reached, and of the degree of obstruction to the circulation thence resulting. In one person, the discoloration may be, under ordinary conditions, scarcely perceptible, and in another, constantly well marked; in one, the physical and moral powers seem to retain their usual energy, even to advanced age, and in another, these powers are always feeble, and life fails at an early period. In the one case, we may suppose that the abnormal condition is such, that the circulation meets with little obstruction in the heart under ordinary circumstances, and that the mixture of the venous and arterial blood occurs only under extraordinary circumstances; and in the other, that the physical defect causes great obstruction to the circulation, and is constantly in action. Where the

physical defects are considerable, life seldom is prolonged.

Symptoms of pulmonary obstructions, and, in fact, also, of congestions of the organs of the abdomen and brain, are frequently observed as attendants of the cyanosis. The pulmonary affections show themselves under the forms of catarrh, hæmorrhage, and bronchorrhœa. The attacks of dyspnœa come on generally in paroxysms, and are not constant. The heart is also enlarged, and its impulse rapid and extensive, and its palpitations violent; faintings, convulsions, and, occasionally, sopor and partial coma indicate the effects of the disease upon the brain.

The *physical signs* must necessarily correspond to, and therefore vary with, the physical defects which have produced the cyanosis; they present to us nothing specially diagnostic of the nature of those defects. Hypertrophy, with dilatation of the right ventricle of the heart, is almost always present, the thickness of its walls being sometimes greater than that of the left ventricle. A systolic bruit is often heard about the base of the heart, and chiefly in the situation of the pulmonary artery, and occasionally a thrill is perceptible at the same part—both the bruit and thrill probably arising in such case from the constriction of the pulmonary artery's orifice. It is possible, also, that a bruit may arise from the passage of the blood through an intra-ventricular

opening or through the foramen ovale.* Bruits may also be present, arising from constriction of the arterial and auriculo-ventricular orifices, or from a defective condition of the valves of the pulmonary artery.

Treatment.—On this head there is little to be said. Inasmuch as the cause of the disease is irremediable, all we can attempt is, by prophylactic treatment, to endeavour to ward off the affections of the lungs and the brain, &c., above described, which result from the physical defect provoking them. The patient should therefore avoid over-exertion, muscular and mental (which, from the nature of the complaint, he is instinctively inclined to do); he should be strictly cautious in his diet, and attentive to the ordinary hygienic rules regarding clothing, food, and exercise. His diet should be nutritious; and he should be carefully guarded against the effects of cold and sudden changes of temperature. The surface of the body, as being a compensating source of aëration of the blood, should be kept in a healthy condition of secretion. The chief treatment during the paroxysms will be to remove the immediate cause which excited them. When convulsions occur, one or two leeches may be applied to the temples; but care must be taken that the bleeding is not too abundant, as it

* See case recorded by myself in *Pathological Journal*, vol. viii. p. 142.

is apt to be in cases of cyanosis, on account of the enlarged condition of the capillaries. The state of the bowels also should be always duly regulated.

CHAPTER XIX.

ATROPHY OF THE HEART.—ITS DISPLACEMENTS.

ATROPHY.—ITS NATURE.—CAUSES.—DISPLACEMENT.—VARIETIES.—CAUSES.—SIGNS.

Atrophy of the Heart.—This condition of the heart consists mainly in a diminution of its muscular tissue. Like the voluntary muscles, the muscles of the heart, and, secondarily, its nerves and arteries, diminish in size, and necessarily also in power, when long subjected to causes which produce general wasting of the body. The valves of the heart also become, under similar conditions, thin, contracted, and atrophied.

The fibres in a wasted muscle, as Mr. Paget tells us (Lect. 1. 117), appear almost perfectly healthy: "they are rather paler and softer, and more disposed to be tortuous, than in the natural state," but their tranverse striæ and other characteristics are well marked.

Atrophy of the heart is observed in connexion with wasting dieases, as in phthisis, diabetes, cancer,

dysentery, &c. And in such cases the diminution of all parts of the organ goes on equally—the cavities as well as the walls becoming smaller. Here there is exactly an opposite condition to that of hypertrophy of the heart. In consequence of there being less blood in the body, less force is required to force it on through the arteries, and so the heart wastes and diminishes in size, accommodating itself to the smaller amount of work required of it. The heart of an adult may thus be reduced from nine to five ounces; thus diminishing instead of increasing with advancing years, as the rule is.

This condition of the heart cannot be regarded as a disease, but merely as one of the consequences which result from general wasting of the body. There are no auscultatory signs pathognomonic of atrophy of the heart; the diminished area of percussion-dulness over the præcordial region is manifestly a sign of little worth. The pulse at the wrist, and the heart's impulse, will naturally be weak; and we may guess at the atrophied condition of the heart from the nature and duration of the wasting disease.

The *treatment* of atrophy of the heart has evidently no direct reference to the organ itself. It is merged in, and in fact is the same as, the treatment of the particular wasting disease which has produced the atrophy.

Displacements in the Heart.—The mode of attach-

ment of the heart allows considerable freedom to its movements, and permits of its being readily displaced from its natural position in the thorax, by pressure exerted upon its outer surface. It may be displaced towards either side; and it may be forced upwards or downwards. The most common cause of its lateral displacement is pleuritic effusion. Effusion of whatever nature, occupying the cavity of one of the pleuræ, compresses the lung, and pushes the heart towards the opposite side of the thorax. Displacement, however, towards the right side appears more readily effected by effusion into the left pleura, than is displacement to the left by effusion into the right pleural cavity. The anatomical relations of the heart explain the reason of this.

Simple hydrothorax rarely produces displacement; for the fluid being equally collected in the two pleuræ, presses alike on either side of the heart, and so retains the organ in its proper site. As occasional causes of its displacements in different directions may be mentioned—solid and fluid tumours, aneurisms, extensive emphysema, and pericardial effusions.

The heart is forced upwards in ascites; and occasionally by enlargement of the liver and of other abdominal organs. It is, also, not unfrequently displaced in women during the latter period of pregnancy. Various diseased conditions of the lungs, likewise, alter its position. In cases of pleu-

risy, for instance, of the right side of the thorax, the fluid having been absorbed, and the lung in the affected side remaining permanently compressed, the heart has been found lying in the right side. And so, again, it has been found lying unnaturally high in the thorax, in consequence of the diminution of the upper part of one of the lungs, resulting from the cicatrization and contraction of a large tubercular cavity. The heart, it has been said, in such cases, is drawn towards the contracted portion of lung. It seems, however, much more probable, that the enlargement of the healthy lung—which naturally in such cases becomes unduly developed to compensate for the loss of the spoilt lung—is the most efficient cause which operates in producing the displacement of the heart, by pushing the organ towards the contracted part of the lung.

The most common cause of the displacement of the heart downwards is double emphysema. Single emphysema—of one lung only—is a very rare affection, and would naturally tend to push the heart laterally. When displaced downwards, the heart is felt beating with unusual force at the epigastrium. But it must be remembered, in judging of this sign, that chronic emphysema of the lungs, and enlargement of the right side of the heart, are very constantly associated together, the latter being the ordinary consequence of the obstruction to the pulmonary circulation which is produced by the emphysema. And inasmuch

as hypertrophy of the right side of the heart of itself occasions epigastric pulsation, we have an explanation of the pulsation felt at the epigastrium in emphysema of the lungs, independently of any displacement of the heart. It is needless to enumerate the possible displacements of the heart through pressure of morbid growths.

The displacements of the heart very rarely cause any alteration in its sounds;* but it is conceivable, that such an altered direction may be thereby given to its arterial trunks, as to occasion a bruit so long as the displacement lasts. Such a case has, indeed, been related by Dr. Walshe. "I once found," he says (Diseases of Heart and Lungs, p. 357), "for many successive days during the height of left pleural effusion, both sounds of the heart, which was pushed to the right of the sternum, more or less masked by blowing murmurs; these murmurs, when the heart was restored, or very nearly restored to its natural position, almost completely disappeared."

The sounds of the heart will naturally be heard loudest, and its impulse be felt strongest, at that part of the thoracic walls beneath which it lies; and thus, by the aid of these signs, and by the presence of a dull percussion-sound where, in a normal

* I have at this moment under observation a case in which, through enlargement of the liver and ascites, the heart is so pushed upwards that its apex beats immediately behind the nipple. Yet its sounds are clear and good.

condition of parts, the sound should be clear, and by the absence of the normal cardiac signs, we are enabled to ascertain the fact of the displacement and its direction.

In considering these signs, however, it must not be forgotten, that when the heart is much enlarged, the hypertrophy will of itself cause an apparent displacement of the heart. Thus its apex may reach far away to the left of the nipple, and be felt beating in the left lateral thoracic region—the heart, in such case, taking a transverse direction, simply by reason of its hypertrophy.

It is difficult, if not impossible, to calculate the effects produced on the circulation and respiration, through displacements of the heart; for the various causes—the pleuritic effusion, or the emphysema, for example — which produce the displacements, must in all cases of themselves create disturbances in those functions. The effects of the displacements, and of their causes, are thus inextricably mixed up together. *A priori* we might reasonably argue, that displacement of the heart would interfere with the integrity of its movements, and consequently that it would in a greater or less degree add to the embarrassment which the circulation and respiration already suffer, through the influence of the cause which produced the displacement. It is, however, surprising to find, in practice, how greatly the heart may be pressed from its natural site, with-

out apparently adding, in any marked manner, to the difficulties of the circulation or the respiration ; and this, too, even when the displacement has taken place suddenly. The freedom of the circulation is very striking in certain cases of chronic empyema, in which the change has taken place slowly. In such cases the heart adapts itself gradually to the lateral pressure ; and besides this its duties are diminished, because the quantity of blood circulating through the lessened capacity of the aërating surface of the lungs is proportionably diminished.

The treatment of these displacements is, of course, the treatment of the particular morbid conditions which produce them, and will therefore not occupy our attention here.

CHAPTER XX.

FUNCTIONAL DISORDERS OF THE HEART.

SYMPTOMS.—DIAGNOSIS.—TREATMENT.—PROGNOSIS.

Functional Disorders of the Heart.—Under this head I include those cardiac derangements which are not associated with organic disease of the heart or arteries. They are very common, depending upon a variety of causes, and occur in both sexes, and at different periods of life.

In women we observe functional cardiac disorders chiefly in connexion with derangement of the uterine function, and particularly at the important climacteric periods of their life, viz., at the commencement and at the cessation of the catamenial discharge. Like most of the functional disorders of females, their occurrence is especially noticeable in coincidence with hysterical symptoms.

In men functional cardiac disorders prevail most frequently during the youthful and adult periods of life. In early life they appear for the most part to be associated with, or to result from, indulgence in

various vices and excesses; amongst which may be prominently mentioned masturbation, tobacco-smoking, spirit-drinking, and tea-drinking. The palpitations in such cases are, of course, only one symptom of the general enfeeblement of the system engendered by the vicious habits and excesses here referred to. I have seen cases of this kind where the severity of the palpitations and the presence of the anæmic murmur have led to the diagnosis of organic disease of the heart. Over-mental exertion also, at this period of life, associated with dyspeptic symptoms, is doubtless one of the promoting causes of palpitations, a fact of which probably most medical students have had personal experience at one period of their lives.

In adult life, functional cardiac affections in men often assume a very important character, and demand a more careful consideration than is, I believe, usually bestowed upon them. They are met with chiefly among professional men and men of studious habits, whose minds have been overwrought, and stomachs overloaded, and whose business is of a sedentary sort. And they are also observed in the overworked and ill-fed artisan.

The palpitations present themselves to us in association with a series of obscure and often very painful symptoms, causing to the subject of them both great mental anxiety as well as bodily suffering. Evidently, their original source is to be found in the exigencies required by the artificial state of society

in which men are now-a-days called upon to play their part in large cities and elsewhere. The unnatural conditions to which men are thus forced to submit, the strain upon their minds, and the irregularities practised upon their bodies at a time when, by reason of nervous exhaustion, the organs are least capable of combating them, are the promoting causes of these affections. They are signs of weakness, and of weakness connected in an especial manner with derangement of the digestive functions. It will, in fact, invariably be found, that one or other or several of the multifarious items which are classified under the head of indigestion, are present in connexion with the cardiac symptoms. Or, more properly speaking, the cardiac symptoms are really one of the indications of derangement of the assimilative functions, only in this case the most prominent indication.

Patients, thus affected, suffer from a variety of sensations which are simply disagreeable and annoying, or more or less positively painful, and all having reference directly to the heart. They complain of palpitations, a sinking, a fulness, a fluttering, a tickling, a creeping sensation at the præcordia; or there is a feeling of deep oppression or want of breath, as of something constantly gnawing at the heart, or of a darting, stabbing there; and with these are associated lowness of spirits, great anxiety, a dread of some impending danger, and continued

morbid attention to their supposed disease of the heart. It is curious, indeed, to note the pertinacity with which patients, under such conditions, will often adhere to the belief that they are suffering from organic disease of the heart; and this is more striking when compared with the well-known fact, that patients, who are really suffering from organic heart-disease, very frequently, in detailing their symptoms, make no allusion whatever to the real seat of all their sufferings.

The symptoms above spoken of are veritable sources of suffering to the patient; and however sure the physician may be that the subject of them has no organic disease of the heart, he must not lightly pass them over as the airy nothings of hypochondriacal fancy. Besides this we must remember, that in some of these cases, and especially at advanced periods of life, there may actually be a *primum mobile* of disorder located in the heart, viz., fatty degeneration of its muscular structure. Some of the symptoms complained of are quite compatible with—indeed are indicative of—such a condition of the heart; and, as we well know, the absence of what are called the physical, *i. e.* the auscultatory, signs of organic disease, are no proof of the non-existence of fatty degeneration of the organ.

The treatment to be adopted in cases of this nature is clear enough. Having satisfied ourselves that the disordered condition of the nervous and digestive

functions are unassociated with any organic disease (as we usually understand the term), we must, as far as possible, withdraw the patient from the influence of those causes which were the original excitors of his derangements, and are still in operation. I need not say what these are in the cases of the functional cardiac disorders, as above referred to, observed in youth and in the female sex; nor need I linger to say what their appropriate treatment should be. But I will add a few words in reference to the management of these affections as they occur in adults. Here they are more closely and manifestly connected with derangements of the digestive organs, as is shown by their frequent occurrence in connexion with gouty and severe dyspeptic disorders, and in those whose mental energies are over-taxed, and whose mode of dieting (as usually happens) is, coincidently, irregular.

In the majority of cases we may at once relieve his chief source of anxiety by assuring the patient of his immunity from organic disease of the heart; though, as I have already said, we shall often find it no easy task to make him believe in the correctness of our diagnosis. I have known a patient of this kind have much greater confidence in the opinion of a doctor, who certified to him that his heart was organically diseased, than that of two physicians of note, who positively assured him that it was quite healthy. We are also almost always able to give

great relief to the bodily as well as the mental sufferings of the patient, by careful application to his case of the ordinary rules for proper regulation of enfeebled organs. The first and simplest rule is to subtract the patient from the evil and unnatural ways of life which have engendered the abnormal state of body; and to explain to him how needful it is that he should treat his body as a machine that has been overworked, and has got out of gear and requires repair. The ignorance of patients as to the true cause of their disorders is surprising enough. Knowing well and admitting the fact of the mental and bodily irregularities to which they have been long subjected, they do not refer to these as the true cause, but to some immediate and trivial accident of yesterday, and they will go to their physician expecting a sudden and a quick cure. They wish to swallow a drug, and then expect that their malady will fly away and themselves be left at rest.

The patient forgets that his ill condition is a chronic affair, that it was not brought about by any single excess, but that it is the accumulated sum of a series of smaller irregularities, which have for a long time been operating on his body, and gradually weakening and undermining its vital forces. The organs (unfortunately, perhaps) do not at first complain of these self-inflicted irregularities which man's reason should teach him to avoid; but, with an admirable adaptability to the exigency, submit to

them for a season, and strive by extra exertion to meet the undue strain. The day of reckoning, however, comes at last, and the over-taxed organs are then found unequal even to their ordinary and natural amount of work. All this it is necessary to explain to the patient, who is surprised that a quick cure cannot be afforded him. He must be taught that a true cure is only to be obtained by a removal of the original sin which he has committed against his body. The old blue pill and black draught system may give immediate and temporary relief; but if a change of the habits of living be not also practised by the patient, the pill and the draught become merely a mockery and a snare. The surprising success of that delusion, homœopathy, is to be sought in an acknowledgment of this plain fact.

It is of course impossible to lay down any special rules of treatment which can be adapted to every case, nor need I mention the thousand promoting causes of this nervous and dyspeptic condition of body. Careful dieting and abstinence from the practice of the causes of the disorder are the main items of the chronic cure required. As secondary aids we may employ various medicinal agencies, as succedanea, to remove the immediate distress, and for the purpose of assisting the *vis vitæ* in restoring the weakened organs, if so it may be, to their original state of power. For this purpose the tonics,

steel and mineral acids, and opium to relieve irritability and nervousness, are our best helps.

Our diagnosis of the absence of organic cardiac disease in these cases is, of course, founded upon the fact of the absence of all the signs and symptoms already described, which are indicative of organic disease. Our prognosis is favourable when the hygienic rules necessary for the recovery of health are adopted and rigidly adhered to. When, however, the patient has been long a sufferer in this way, is somewhat advanced in years, and has long continued in the habit of over-taxing his mind and bodily organs, and if he has at length become incapacitated for work, and has withdrawn from occupation, a favourable termination of his case is hardly to be expected. Under such circumstances he broods over his misfortunes until his painful sensations become the sole object of his thought, and is then apt to settle down into the state of an undeniable hypochondriac.

I am informed by Mr. R. Martin, that the pulse of Europeans who are beginning to feel the effects of long residence in India, often becomes irregular and intermittent; and that this condition of it may last for some time after the individuals, in whom it is observed, have been removed from the climate and the influences which have produced the irregularity. The fact is, I think, not generally known, but is well worthy the attention of those who may be called upon to attend patients who have

resided in the East. This condition of the pulse must clearly be attributed to functional disorders of the heart, and results apparently from the depressive influences of a tropical climate on the European constitution. This is evident from the circumstance also stated by Mr. Martin, that the irregularity of the pulse referred to usually disappears when the person has been for some time removed from the action of those influences which occasioned it.

I may also mention, under this head, that we occasionally meet with persons in whom there is a distinct intermission of pulse, but who are nevertheless in apparently perfectly good health, and have never suffered from, nor present any signs of, cardiac affection. I believe that this state of the pulse is merely temporary, and may be traced to over-exertion of the mind, and other depressing influences. It is, however, a difficulty which the medical officers of Assurance Societies have occasionally to meet.

CHAPTER XXI.

DISEASES OF THE AORTA.

ANEURISMS.—PATHOLOGY.—CONSEQUENCES WHICH RESULT FROM THEM.—ANEURISMS OF THE THORACIC AORTA.—THEIR SITUATION AND FORM.—THEIR EFFECTS UPON THE PARTS AROUND THEM.—THEIR DIAGNOSIS.—PHYSICAL SIGNS.—AUSCULTATORY SIGNS.—GENERAL SYMPTOMS.—DYSPHAGIA.—PAIN.—PRESSURE UPON THE PARTS AROUND.—GENERAL HEALTH.—COURSE.—PROGNOSIS.—TREATMENT.—OBLITERATION OF THE AORTA.—SIGNS.

THE present state of our knowledge on the subject of inflammation of the aorta enables us to say little which can be turned to any practical account concerning it. Its existence as a disease is hardly demonstrable during life, and the signs which it leaves after death are involved in much obscurity.

The pathological changes of the aorta and the arteries which demand the attention of the physician are those which arise in connexion with, and as consequences of atheromatous, albuminous, and calcareous degenerations of their coats. These changes are very frequently met with in the aorta, the conditions under which they occur being similar to those which give rise to like abnormal deposits in and

around the valves of the heart, and of whose pathology I have already treated. Such deposits sometimes occupy large portions of the vessel, spreading over and lining its inner surface; and when the deposit is of a calcareous nature, may even convert the vessel into something approaching to a tube with unyielding walls.

The coats of the aorta are so frequently found more or less degenerated at advanced periods of life, that some pathologists have regarded the change rather as a natural mode of senile decay of the tissues, than as the consequence of active disease.

All these different deposits, whether the consequences of degeneration or of inflammation, have the effect of injuring the proper tissues which form the vessel, and of destroying or diminishing their natural, *i. e.* their vital and physical properties. The elasticity and contractility of the vessel are injured in two ways: by the presence of the abnormal deposit itself; and by the injury which the different structures of the vessel have undergone, as respects their innervation and nutrition, in consequence of, and during the progress of, the deposition.*

* The division of these pathological changes of the arterial coats into half-a-dozen different kinds may be interesting to the essential pathologist, but is of no practical service to the physician. I shall, therefore, merely enumerate the following forms:— Fatty degeneration, consisting of opaque, whitish or yellowish, and slightly elevated spots, which are found, microscopically, to consist of fine fatty molecules, situated in the elements of the

Dilatation of the Aorta is one of the ordinary consequences which result from such alterations of its coats. The enlargement of the calibre of the vessel is particularly observed in the first and second portions of its arch, which are the most directly and immediately exposed to the dilating force of the blood thrown into it during the heart's systole, and are also—probably for this reason—more subject to disease than other parts of the aorta. The dilatation of the vessel usually affects its whole calibre, but is most marked, perhaps, in the upper part of the arch, where the current of blood impinges with greatest force. Sometimes the vessel is weaker, and so more rapidly yields, at some points than at others; and in this way little pouches are formed, which project from the external surface of the dilated aorta. The large arterial trunks which rise at a right angle from the first part of such a dilated aorta, almost always participate in the dilatation, but the left subclavian, which passes off at an acute angle, does not do so, at least to so great an extent.

inner coat of the artery; albuminous degeneration of the inner coat, mostly also in the form of little spots, or more or less diffused; cartilaginous deposits, forming larger or smaller spots and elevations of the inner coat, irregular, opaque, whitish, thick, like consolidated albumen or cartilage. It is probably an advanced condition of the albuminous degeneration. The other forms are the atheromatous and calcareous; and these indeed are but advanced conditions, or rather retrograde developments of the albuminous and cartilaginous forms. (See Bamberger for a fuller account, *Lehrbuch der Krankheiten des Herzens.*)

A certain degree of obstruction to the circulation necessarily results from this hardened condition and dilatation of the aorta; and besides this, the whole office of forcing the blood onwards is now thrown upon the heart. Hence arise hypertrophy and dilatation of the heart. In health, the contractility, as well as the elasticity of the aorta, aid in carrying on the circulation. The aorta is distended by the blood which is thrown into it during the systole of the heart, and then, during the diastole of the organ, its contractile and elastic forces are brought into play. Re-acting upon the blood within it, the vessel forces the fluid onwards in the direction of the circulation, the backward flow of blood into the ventricle being prevented by the closure of the semilunar valves.*

Contraction of the healthy aorta is sometimes observed in certain cardiac and pulmonary diseases;

* The *elasticity* of the arteries appears to be simply a physical property of their tissues, still existing, and in a very marked degree, even after their death. Their *contractility*, however, is a vital property, and results from the action of the muscular elements which are associated with the elastic fibres. It is often forgotten, in arguing upon the circulation, that the flow of blood through the arteries is impeded by their loss of contractility, as well as their loss of elasticity. It has long been an admitted fact in physics, that the rigidity or dilatability of the walls of a tube has very little influence over the quantity of fluid which passes through, *provided the current is determined by a constant pressure.* But when the impelling force is intermittent, then the elasticity of the walls of the tube aids considerably in augmenting the flow. (Milne-Edwards, *Leçons de Physiologie.*) Moreover, the loss of distensibility of the vessel increases the amount

in those, for instance, in which a due amount of blood is not thrown into the vessel at each systole of the heart. This condition occurs when there is obstruction to the entrance of the blood into the left ventricle, or to the pulmonary circulation in chronic diseases, &c. The pressure, also, from without, of tumours, abscesses, &c., situated in the vicinity of the vessel, may so compress its ascending portion as to reduce the calibre of the aorta. Its calibre has also been found diminished by an excessive growth of calcareous matter projecting into it, and occasioning portions of the fibrin of the blood to become entangled about and deposited upon its roughened surfaces.

Hypertrophy and dilatation of the heart are often associated with these diseased conditions of the aorta, but there are no particular physical signs or symptoms which can be considered as pathognomonic of them. Dulness on percussion over the ascending aorta, as a sign of dilatation of the vessel, is of very little value; for the detection of an abnormal dulness in such case is scarcely possible, unless the enlargement of the aorta is very great.

Roughnesses on the inner surface of the aorta, when considerable, will occasion arterial bruits; but it must be remembered, that atheromatous or cal-

of friction; because then the rapidity of the current of blood is increased, and friction increases in proportion to the squares of the rapidity of the current, and this loss, therefore, of distensibility, causes in another way an impediment to the circulation.

careous degenerations very rarely affect to any extent the first portion of the aorta, without likewise involving more or less its valvular apparatus, and so giving rise to systolic or diastolic bruits. Occasionally, however, a systolic bruit is heard louder about the upper part of the sternum than it is over the midsternum; that is, near the aortic valves. Such a bruit may be found associated with dilatation of the aorta and with roughness of its internal surface, without any coexisting disease of the aortic valves or the aortic openings.

Aneurism of the Thoracic Aorta.—All enlargements of the aorta are, in one sense, aneurismatic enlargements, and therefore dilatation of the aorta, as above described, might be included under the head. The term is, however, practically used in a more limited sense, and is applied to those cases in which the dilatation of the whole circumference of the aorta is considerable, whether the dilatation be globular or fusiform; and also to all those distinct bulgings or enlargements of the vessel which affect only a part of its circumference.

These different enlargements of the aorta have been divided into a variety of kinds, according to the form and position which they assume in relation to the vessel; but no practical advantage is gained by such subdivision, for aneurisms may arise at any portion of the aorta, though doubtless some parts of it are more frequently affected than others.

The very fact of this enlargement of the vesse conveys with it the idea of imperfection in the con dition of its tissues; and we may, indeed, set it dow as a rule, that the coats of the vessel where such aneurismal swelling appears, cannot be in a norma state. Moreover, the existence of aneurism in any part of the arterial system, usually indicates a dis ordered condition of the vascular system generally throughout the body. The surgeon well knows, that in the case of many of those particular aneurisms which are the objects of his operations, he has to consider not only the mere aneurismal swelling, but also the condition of the affected artery on which he operates and of other parts of the arterial system. The vessel may be diseased at the point where he operates, and there may likewise be aneurisms of other arteries of the body yet undiscovered by him.

Thoracic aneurisms present themselves to us under various forms, and at different parts of the thoracic aorta. Their most common situation is in the ascending and transverse portions of the aorta; much more rarely are they met with in its descending portion. The reason of this probably is, that the parts of the aorta nearest to the heart are more frequently diseased than those at a distance; and that when the aorta is generally and extensively affected, the disease is more advanced in that portion of the vessel which is immediately connected with the heart.

It is not necessary to linger over a description of

the different shapes which aneurismal tumours may assume, for these are evidently accidental circumstances in no way affecting the pathology of the disease. And besides this, with the progress of the disease, and the enlargement of the tumour, the aneurism may undergo considerable change in form, one kind of aneurismal pouch being thus converted into another. I will only repeat, that the swelling may embrace the whole circumference of the tube, and that it may arise at one part only of its walls. Aneurisms, also, vary greatly in size; we meet with them of the size of a nut, and as large as a man's head.

The condition of the walls which constitute the aneurismal tumour—the sac—differs much in different cases. The three coats of the vessel, which in health form its proper tunics, may, though altered in structure, remain unbroken, and form the covering of the tumour; or the inner membrane may be torn or destroyed, and leave the two outer tunics of the vessel to form its walls; or the sac may be constituted of the inner and outer tunics, the middle coat being injured by disease; or the inner and middle coats of the vessel may be destroyed, and the aneurismal sac formed of the outer membrane only; or the outer coat may be destroyed, and the sac consist of the two inner tunics of the vessel; and, lastly, the two inner tunics may be partially or totally ruptured, and the blood escaping therefrom separate

the outer tunic from them, and so produce a large, diffused, and extensively spreading tumour, called dissecting aneurism.

These different divisions of the sac are, however, of little practical import, for it is not always easy to distinguish the anatomical divisions of the sac after death, when the disease is of some standing. As we have already seen, the arterial coats are in such cases almost always much altered from the healthy state, independently of the changes which they have suffered through the progress of the aneurismal swelling; the divisions, therefore, above given, of the constitution of the sac in aneurisms, must not be looked upon as unexceptional, or as of any great service to us in practice.

The contents of the aneurismal sac are either fluid or solid: usually, we find within it layers of coagulated fibrin and fluid blood, the blood of course communicating with that circulating in the vessel itself. Sometimes the layers of coagulated fibrin are so thick as nearly to fill up the sac, and render it solid; and in some rare cases, small aneurismal tumours of the aorta have thus seemed to have been cured. The layers of fibrin are drier and firmer, the nearer they are to the walls of the sac; those lying in contact with the blood are generally loose and soft, and have a shaggy-looking surface. Coagulation of the fibrin within the sac, seems to be favoured by smallness of the aperture of communication which exists between

the vessel and the interior of the sac projecting from it. The walls of the aneurism are, of course, necessarily strengthened by these firm layers of fibrin. Sometimes the walls of the aneurism are in part formed by the condensed structures of the organs upon which the tumour presses in its growth.

Of much more importance to us than the form and constitution of the aneurismal sac, is a knowledge of the effects which it produces on the parts and organs with which it comes in contact during the progress of its growth; for it is from a consideration of these effects that we form our diagnosis, previously, at least, to the moment when physical signs appear, and render the diagnosis more sure. It generally happens, however, that it is only when the aneurism has made considerable progress, and is, in fact, completely established as such, that we discover its existence during life.

Thoracic aneurisms are most frequently met with in the ascending portion and arch of the aorta, and are almost invariably situated on the anterior or lateral surfaces of the vessel. In growing, they naturally tend to enlarge in that direction which affords least opposition to their expansion. When the aneurism is small, it may excite no disturbance in parts around it; and hence it frequently happens that an aneurism is discovered after death, whose existence had never been suspected during life.

It may be said, generally, that the different effects produced by the growth of a thoracic aneurism depend upon the same cause,—namely, the mechanical pressure of the tumour exerted upon the organs and parts around it. This pressure may displace the parts around the tumour; or it may interfere with their functions; or it may destroy and injure their structures; it may excite inflammation in them, and its consequences; and, by pressure upon, and irritation of nerves, give rise to spasmodic and reflex muscular actions, and to great pain.

By pressure on the trachea, or one or other of the bronchi, respiration is interfered with, and cough of an irremediable nature excited. Cough is also, in many cases, the result of irritation of the recurrent nerve, produced through the stretching of it by the tumour. The aneurism not unfrequently perforates the trachea or one of the large bronchial tubes, and sometimes suddenly bursts into them, causing instant death by suffocation. By its pressure upon the lungs, the tumour excites inflammation of, and effusion of lymph into the pulmonary tissues, producing fatal pneumonia and pleurisy: sometimes it destroys the tissue, and gives rise to pulmonary hæmorrhage. Inflammation of the tracheal and bronchial membranes may also be mentioned as direct or indirect results of the presence of the tumour. By its pressure on the œsophagus, difficulty of swallowing is produced, and sometimes even symptoms of œsopha-

geal stricture.* The pressure on the œsophagus may likewise produce ulceration and destruction of a portion of the tube,

The tumour may also press upon the roots of the great arteries which take their rise from the aorta, and partially obstruct the circulation of blood through them, by distorting them and pushing them from their proper places, or causing a deposition of fibrinous plugs within them; and so, also, it may prevent the return of blood to the heart, by obstructing the passage of the blood through the large venous trunks. When the aneurism takes its rise in the aorta near the heart, it may press upon the walls of the cavities of the heart itself, and also force the heart aside from its natural position in the thorax. The thoracic duct may likewise be compressed by pressure of the aneurism. When the tumour enlarges towards the surface of the body, it destroys, by its incessant pressure, the bones and soft parts which form the walls of the chest.

From all which it follows, that thoracic aneurisms may interfere with, or arrest entirely, the functions of the parts with which they come in contact, and may also destroy and disorganize the structures subjected to its pressure.

The heart itself may remain healthy, but gene-

* This is one of the many cases which warn the surgeon of the great consideration required in practising catheterism of the œsophagus.

rally we find it hypertrophied, in cases of aortic aneurism; for the diseased condition of the walls of the aorta, which occasions the aneurism, is of itself sufficient to provoke hypertrophy of the heart, in consequence of the obstruction to the circulation which results from the loss of the elasticity of the coats of the artery. Hypertrophy is also promoted by the impeded pulmonary circulation, which often coexists with the aneurism.

Diagnosis.—The diagnosis of aneurisms of the thoracic aorta is founded on a consideration of the symptoms which are produced by their pressure on neighbouring parts and organs; and also upon the external appearances and stethoscopic signs, which they occasionally present in their advanced stages of growth. The existence of small aneurisms very frequently passes unnoticed during life; and the same is sometimes true even in the case of large aneurisms. The aneurism, indeed, may declare its presence for the first time, when it suddenly destroys life by bursting internally. In such cases the tumour grows in such a direction as not to interfere, by its pressure, with the functions of any neighbouring part. Much obscurity, therefore, often surrounds the diagnosis of thoracic aneurism; and in most cases, it is only when we can feel, and, as it were, see, the tumour—when it has made its way towards the surface of the body, and projecting from beneath the skin, heaves and beats synchronously with the systole

of the heart, that we can positively diagnose its existence.

The signs and symptoms which result from obstructions and injuries of different parts and organs through pressure of the aneurismal tumour are, however, sometimes such as to leave no rational doubt in the mind of the observer as to its nature; and this, too, even prior to the existence of any special auscultatory sign. We have, therefore, to consider the physical signs and the symptoms attending thoracic aneurism.

Physical Signs of Thoracic Aneurism.—The most remarkable sign, when it exists, is that of a prominence—generally of an obtusely conical form—of some part of the thoracic walls. An aneurismal prominence, situated about the right and upper part of the sternum, especially indicates a connexion of the tumour with the ascending portion of the aorta. When the transverse portion of the arch is the seat of the aneurism, then the tumour is developed in a direction upwards towards the top of the sternum, and perhaps rather towards the right of it. Aneurisms situated on the descending portion of the thoracic aorta, rarely give rise to any prominence of the thoracic walls anteriorly; they usually enlarge into that side of the thorax—the left—down which the aorta passes, and sometimes they destroy the ribs and vertebræ, and present themselves behind in the dorsal region; but such development

is rare. These are the general directions which thoracic aneurisms take in the progress of their growth; but there are, of course, many exceptions thereto.

The aneurism, as it advances, not only destroys the soft parts which form the parietes of the chest, but it likewise gradually erodes the bones, by the constant pressure which it exercises upon them. The cartilaginous portions of the thoracic walls, on account of their more elastic and yielding structure, resist destruction longer than the bones. The skin remains long unchanged, but it at last likewise succumbs under the pressure of the advancing tumour: it becomes thin and red, and sometimes ulcerates, and so allows the blood to escape with a gush from the sac. The thinness of the skin and of the walls of the sac, at the prominent part of the tumour, is really, at times, fearful to feel and witness, especially when the pulsation of the aneurism is strong: one cannot but dread that every beat of the heart may cause rupture of the sac. Even under such circumstances, however, the fatal moment may be long averted by judicious management.

Auscultatory Signs.—The sounds heard over the aneurismal tumour vary considerably in different cases; and sometimes no abnormal sound whatever is heard.* This fact is readily explained by the

* According to Dr. Stokes, the presence of a murmur is exceptional in cases of thoracic aneurism; that is, it is more

particular formation of the aneurismal sac and the nature of its contents. In some instances, the mouth of the sac is small, or partially obstructed by fibrinous coagula; in others, it is large and open, permitting the free entrance and exit of the circulating blood. At one time, the sac is almost entirely blocked up by layers of coagulated fibrin, so as to become, as it were, a solid tumour; and at another, its contents are nearly altogether fluid. The position of the tumour, and the direction in which it grows, also materially affect the free passage of blood into and out of it; the tumour may be large and the neck of the sac partially twisted, and so the current of blood into it obstructed. The strength of the heart's action; the position of the opening of the sac relatively to the direction of the current which is forced from the heart; the partially elastic or completely inelastic nature of the walls of the sac; the distance of the tumour from the walls of the chest, are circumstances which necessarily affect the character of the sounds which arise in the aneurismal tumour. Hence also we learn why a murmur may be absent at one period, and present at another, during the progress of the aneurism.

Double and single murmurs and sounds, corresponding closely in time with the systole and diastole of the heart, and resembling those which arise

frequently absent than present. In this, however, my own experience differs from his.

in the heart, may be heard over the aneurismal sac. The systolic murmur may be of various kinds,—soft and blowing, and harsh and grating. The diastolic murmur is much less frequently observed, and is softer than the systolic,—a fact which is explained by the nature of its probable cause, viz., the passage of the blood, during the diastole of the heart (and the *systole* of the aneurism), out of the sac, through its narrow opening, into the aorta. The force which thus expels the blood out of, is less than that which drives it into the sac.

Sometimes the murmur may be produced by the pressure of the sac itself on the aorta, or on one of the large vessels arising from it. And besides this, it is well to remember, that the aortic valves may be themselves coincidentally diseased; and, consequently, that the murmurs heard over the aneurism may be in reality produced at the aortic orifice.

Together with the murmur heard over the tumour, or the supposed seat of the aneurism, the auscultator may feel an impulse through the stethoscope placed over the aneurism, at each systole of the heart—an impulse as strong as, or even stronger than, that felt over the heart. This sign becomes more striking, when at the same time little or no impulse is perceptible over the thoracic walls, between those points where the impulse of the heart and that of the aneurism are felt.

Percussion Sounds.—The percussion sound of an

aneurismal tumour, where it comes in contact with, or projects through, the thoracic walls, is necessarily dull. The extent of the dulness will depend upon the size of the tumour, the amount of its circumference which comes in contact with the thoracic walls, and also upon the condition of the neighbouring parts. It is necessary to remember, that the lung is apt to be consolidated in the neighbourhood of the tumour, in consequence of the inflammation excited in its tissue by the pressure of the tumour upon it. The dull percussion is most indicative when it is found at parts where the sound is clear in health; and so to the right of, and upper part of the sternum: lower down, and to the left of the sternum, the dull sound becomes obscured by the pressure of the heart, &c.

A powerful impulse is communicated to the hand, when laid over the part against which the tumour impinges; and sometimes a distinct vibratory thrill is likewise felt, coincident with the systole of the heart. Also, when the tumour has pressed through the thoracic walls, a back stroke is occasionally perceived, following immediately upon the impulse. When the aneurism is situated at the arch of the aorta, the impulse of the tumour is usually felt most strongly above the notch of the sternum.

Symptoms of Aortic Aneurism.—Thoracic aneurism, as I have already said, may long escape detection, producing neither physical nor general signs; and

may even attain a considerable size without provoking them. Generally, however, symptoms, more or less obscure, early indicate the presence of something abnormal within the thorax.

These symptoms result from the local pressure of the aneurism upon the parts around it. And in this way pain, dyspnœa, cough, hæmoptysis, dysphagia, and alterations of the voice are produced.

The pain suffered by the patient varies in kind and in situation; it has a reference to the position of the tumour, and to the particular nerves which are compressed or irritated by the tumour. A gnawing, rending, constant pain is felt in the back, when the tumour is pressing on the dorsal vertebræ; and radiates in the direction of the distribution of the dorsal nerves. When the nervous plexuses of the neck are involved, the pain reaches down the arms, and is chiefly felt in the neck. Then, again, pain may result from pleurisy excited by the growth of the tumour; from irritation of the intercostal and cutaneous nerves; from periosteal inflammation; and from spasms of the diaphragm caused by irritation of the phrenic nerve. Paroxysms of pain and dyspnœa may also arise from pressure on the trachea, and from stretching of the recurrent laryngeal nerves; and this pain and dyspnœa, when, through the advanced growth of the tumour, the pressure on the bronchi or trachea, and the irritation of the laryngeal

nerve, have become incessant, are often fearful sources of distress to the patient.

Cough, &c.—When the trachea, larynx, and recurrent nerve are compressed by the tumour, the voice is often much altered: it may be weak, hoarse, trembling, husky, and even almost lost. At the same time the breathing is interrupted, and a painful cough of a peculiar dry, ringing character may be present. Such a cough, of an extremely irremediable character, I have known as the only symptom of an aneurism which proved suddenly fatal. *Hæmoptysis* is a much less common symptom than cough and dyspnœa. Pneumonia, congestion, bronchitis, pleurisy, and other pulmonary affections are all occasional accidents attendant upon aneurism, being produced either directly, by the pressure of the tumour on the bronchi or pulmonary tissues; or indirectly, by its interference with the circulation of the blood through the heart and its great vessels.

Dysphagia.—Difficulty of swallowing occurs when the tumour presses upon, or even comes slightly in contact with, the œsophagus. Sometimes the pressure excites ulceration of the œsophagus, and thus, and from rupture of the sac, blood may escape and pass into the stomach, and be thence rejected by vomiting,—producing a false kind of hæmatemesis.

Pressure of the aneurism upon the large venous trunks at the base of the heart occasions congestions and œdematous conditions of the parts, in which

the branches of those veins are distributed, particularly of the upper extremities of the head and neck. Œdema of the lower extremities is rarely observed.*

The *course* which aortic thoracic aneurisms run is, with very rare exceptions, progressive, and their termination fatal. Sometimes the disease seems to pause and linger for a time; and occasionally it would appear that even a complete cure, by occlusion with fibrin, of a small aortic aneurism may take place. Aneurism may destroy life immediately, through rupture of its sac and sudden loss of blood, the blood escaping externally, or into the pleura or the pericardium, into the bronchial tubes, or the œsophagus, or into the pulmonary tissue itself, &c. Or death may result from gradual exhaustion of the system,—the escape of blood being small, and again and again repeated. Aneurism may also prove fatal by exciting pulmonary or pleuritic inflammations,—by pressure upon the trachea and lungs, and upon the œsophagus. Death may also result from inanition, as a consequence of pressure upon the thoracic duct; and from the exhaustion and loss of appetite

* For a more minute account of the symptoms of aortic aneurism, and of its general history, I must refer the reader to Dr. Sibson's account of the "Aorta and the Aneurisms of the Aorta," in his "Medical Anatomy." The reader will there find the most complete and valuable statistical data on this subject which, I believe, have ever been brought together and reasoned upon.

caused by the severe pain, restlessness, and nervous irritation which so often accompany the disease. The most common cause of death, however, is rupture of the aneurismal sac.

Diagnosis.—When the most prominent of the signs and symptoms related above are present in any given case, the diagnosis of an aortic thoracic aneurism becomes easy. A pulsating tumour occupying the position of the first portion of the aorta, dull to percussion, yielding a double or single murmur, and communicating a thrill to the hand; united with difficulty of respiration and deglutition; and with cough, pain, and change of voice—leave little room for doubt as to its nature. It is well, however, to know, that there are diseases which stimulate aneurism, and sometimes in a remarkable manner. Pulsation, for instance, may be communicated to a tumour not aneurismal, by the beat of a healthy subjacent aorta; and such tumour may press upon the aorta and give rise to murmurs, and it may press upon the trachea and œsophagus, and cause difficulty of respiration and deglutition. Also, there are morbid conditions, as well as that of aneurism, which may cause dull percussion about the aortic arch and along the course of the descending aorta in the back. In all these cases, however, the attendant circumstances, and careful study of the history of the disease, generally enable us to fix the true nature of the morbid condition, which thus stimulates aneu-

rism. But, as we have seen, there are aneurisms which present no symptoms or auscultatory signs, by which their presence can be recognised during life; and then, again, there are aneurisms which, according to their size and situation and direction of growth, present symptoms indicative only obscurely of their existence. In such cases our diagnosis must necessarily be hesitating; and will be more or less conclusive according to the degree of clearness of the symptoms.*

Treatment.—From what has been said of the course of an aortic aneurism, it follows, that we cannot hope by any special treatment to effect a cure of the disease, but by careful management, the physician may prolong the life of his patient, and alleviate the great sufferings which so frequently accompany thoracic aneurism. The diet should be nourishing and yet not stimulating; and excitement, mental as well as bodily, most carefully avoided. In other words, the heart should be maintained in a quiet state, and the powers of life duly supported. How often has it happened, that the lives of persons suffering from aneurism have been cut short by

* Dr. Gairdner, of Edinburgh, has endeavoured to show that continuous, though moderate hæmoptysis, accompanied by stridulous breathing, is diagnostic of aneurism, when there is no coexisting disease of the heart or lungs. His opinion is strengthened by the large statistics of Dr. Sibson, who found that stridulous breathing and hæmoptysis were present in one-fourth of the cases of aneurism collected by him.

sudden rupture of the aneurismal sac, resulting from violent action of the heart, brought on by passion or some great bodily effort!

No one, now-a-days, adopts the method of treatment recommended by Valsalva: the venesections he advised for the cure of aneurism, are on all hands admitted to be most injurious and dangerous. In sacculated aneurisms our desire naturally is, to induce and promote the deposition of fibrin, for, by the layers of fibrin deposited in the aneurism, the sac is strengthened, and an attempt at cure is so far begun; but it is worse than useless to attempt to produce such coagulation of the blood by venesection. The state of anæmia which is produced by repeated bleedings, is just the condition we should least desire to see the patient fall into, being that which would give least hopes of his ultimate recovery. In a strong, vigorous person, in whom the heart's action is excited, an occasional bleeding, done for the express purpose of tranquillising the circulation, and relieving pain or pulmonary or cardiac congestions, may be safely resorted to. A few leeches also now and then applied over the aneurism often give considerable temporary relief to the patient.

Those medicines, also, which tend to tranquillise the circulation and soothe the sufferings of the patient—digitalis and opium, hydrocyanic acid, and conium—may be resorted to as required. The bowels should be kept free by laxatives; and constipation

particularly avoided, on account of the straining at stool which it occasions. Gallic acid and acetate of lead have been highly recommended, as promoting coagulation of the blood; but their effects in this way are far from certain. Injections of iodine into the tumour, and the application of moxas on the skin over it, are remedies not likely to find much favour in this country.

Local applications give great relief to the patient: cold applications, and ice laid on the tumour, sometimes afford much comfort, and may possibly promote coagulation within the sac. When the tumour is at all prominent, it is requisite to apply over it some mechanical covering; on the one hand, to protect it from external injury, and on the other, to support and restrain the progress of the tumour.

Obliteration of the Aorta is, I need hardly say, a pathological condition of the vessel which is very rarely met with. But the peculiar phenomena to which it gives rise should be known to the medical observer. Dr. Walshe has given a most excellent description, excellently reasoned on, of a diagnosed case of this kind (*Medical Times*, Oct. 17, 1857). And in the *Medical Times*, May 16, 1857, will be found details of a case, which was found in a dissecting-room. No history was attached to it. There was, however, nothing to show that the abnormity caused death. The obliteration was at the commencement of the descending aorta, just below

the junction of the ductus arteriosus. The ends of the aorta were united by a ligamentous cord half an inch long.

Skoda, in the *Woch. der Zeit. der K. K. G. zu Wien.* Nov. 1855, gives the following account of this disease :—

Obliteration of thoracic aorta always takes place at the part at and between the entrance of the ductus Botalli and the origin of the left subclavian artery.—*Diagnosis.* A peculiar vibration coincident with the heart's impulse (Schwirren) may be heard over the greater part of the chest, and felt by the hand; it arises from dilatation of the thoracic arteries. The beat of the crural arteries is very weak, and no palpitation is felt in the abdominal aorta. The collateral circulation is carried on by the branches of the subclavian arteries; a large amount of blood passes from the anterior to the posterior intercostals, and thus reaches the abdominal aorta. The inferior extremities probably also receive a supply through the anastomosis of the superior with the inferior epigastric aorta. There is no cyanosis. Murmurs may be heard near the heart, and arise from arteries in its vicinity, not in the heart itself. Nutrition in this case was unimpaired, showing that impaired nutrition does not depend solely upon impeded circulation. The obliteration arose during fœtal life and not as a consequence of arteritis, which would rather occasion

distension of the arterial coats. Error in diagnosis can only be avoided by ascertaining the coincidence or otherwise of the bruit with the heart's movements. Skoda also remarks, that a similar case had some years before occurred to him in his Clinique, in the person of a man who died afterwards of pneumonia.

APPENDIX I.

THE USES OF VENESECTION.

I BELIEVE, that the chief cause of the differences of opinion entertained by physicians concerning the effects of blood-letting in disease, is to be sought in the fact, that they are not sufficiently agreed as to the mode of action and the uses of remedy. I have elsewhere endeavoured to point out what I consider to be the use of venesection; and I venture to think, that the explanation which I have given reconciles the differences of opinion above alluded to. I will here shortly repeat the argument.

It is necessary, in the first place, to draw a complete distinction between the direct abstraction of blood from an inflamed part, and the effects of venesection upon it. Leeches applied to an inflamed joint almost invaribly reduce the chief characteristics of the inflammation,—the pain, heat, redness, and swelling; but venesection has no such influence over the inflammation, even though ten times more blood be taken from the body,—nor any effects at all, except such as always result from large abstractions of blood, viz.: syncope and temporary anæsthesia. Now if these facts be true of *external* inflammations, they must, by strict analogy, be equally true of internal inflammations; and, consequently, *local* abstraction of blood should be of service in all those cases of internal inflammation in which there is a distinct vascular connexion between the skin and the inflamed parts beneath. And thus, indeed, we find it to be, as in pleurisy and peritonitis, &c. So, again, venesection, by analogy, should have no *directly* beneficial influence over

internal inflammations; and I maintain that there is no proof that it has.

How comes it, then, that the wisest of our profession have in all ages deemed the remedy so useful an one in internal inflammations? Are we to believe they have all been mistaken? Most assuredly not.

Bleeding is often of the highest service, but it has no directly beneficial influence over the inflammation, as they thought it had. It acts by relieving the pulmonary and cardiac congestions, which arise incidentally and as consequences of the inflammation. Hence, then, bleeding should be of service only in those inflammations which occasion congestions of the heart and lungs; and this we find (at least, now-a-days) to be actually the case. Indeed, it is a fact well worthy of note, that the chief battle-field in which the blood-letting controversy has always been fought is pneumonia—the disease, *præ aliis*, in which the congestions of heart and lungs here spoken of, arising from the interrupted respiratory function, are most readily excited.

As a corollary of these views of the indirect uses of venesection, it follows that it is an excellent remedy in all those diseases, whatever their nature, which occasion a congested condition of heart and lungs. It also follows that a very different signification from that generally adopted must be attached to the remedy, and that the remedy is now-a-days much less frequently resorted to than sane therapeutics require. The change-of-type theory of disease in no way affects the position here assumed as to the uses of bleeding. Whether that theory be true or false, the action of bleeding in disease, be it good or be it bad, must be physiologically the same. That theory can only affect the necessity or otherwise of our resorting to the remedy in any given case; it cannot alter its mode of action.

APPENDIX II.

FIBRINOUS ARTERIAL CLOTS.

A FEMALE, aged 50, the subject of the following history, came into St. Mary's Hospital on the 4th instant. She walked into the Hospital, and gave the following account of her previous condition; thereby showing, that at this time there existed no paralysis either of sensation or of motion. She stated, that when very young she had suffered from some severe affection of the chest, and had ever since from time to time been in an ailing condition. The chief symptoms which troubled her were palpitations, cough, and a feeling of constriction of the chest. From the circumstance that old organized adhesions of the pericardial surfaces were found after death, and from the nature of the symptoms here mentioned, we may presume that this attack was one of pericarditis. Latterly these symptoms had increased in severity, and had incapacitated her for performing any hard work; in addition, she had also frequently had attacks of faintness and giddiness, and particularly in cold weather and on exertion. Occasionally, indeed, when out walking in the street, she had been forced to cling to the walls or railings for support; and on account of her staggering gait, she had been several times unjustly accused of being drunk. These symptoms increasing in severity, and her breathing in particular being very difficult, she sought relief in the Hospital.

On her admission, her lips were blue, her breathing very hurried, and the veins of her neck were much swollen. It was also remarked that the pulse at the right wrist was

imperceptible. At this time she was perfectly sensible, and had perfect command over her muscles. She took her dinner, and went to bed; and in about an hour afterwards was found lying low in the bed, on her back and insensible. Her right arm and leg were completely paralysed, presenting not the smallest rigidity when raised or moved. The pupils of both eyes were firmly contracted and immovable. The pulse at the right wrist was not perceptible, neither was any pulse to be felt in the arteries on the right side of the head and neck; I fancied, however, that I felt a very slight pulsation in the upper part of the right brachial artery. Occasionally, during her heavy breathing, the right cheek was puffed out.

Loud râles were heard over the front of the thorax; the breathing was very noisy, so that it was not easy to make a very careful stethoscopic examination of the heart's sounds. I satisfied myself, however, of the existence of a diastolic aortic murmur—a bruit, coincident with the diastole of the heart, being audible from the middle of the sternum down to its lower edge.

The arrest of the circulation through the vessels supplied by the arteria innominata, the diseased condition of the aortic valves indicated by the bruit, and the suddenness of the paralysis, caused me at this time to hazard the conjecture that a clot of fibrin had suddenly arrested the circulation through some one of the cerebral arteries—a conjecture which was fully justified subsequently.

The difficulty of breathing gradually increased, the paralysis and other symptoms remaining the same, so that the patient died about sixty hours after the first occurrence of the paralysis.

Post-mortem examination.—When the body was examined, the limbs of both sides were found of equal rigidity; there was no œdema, and but little fat on the surface; the superficial tissues were remarkably dry and bloodless. In both pleuræ there was a considerable quantity of serous effusion; the lower lobe of the left lung was thereby compressed and

rendered nearly solid; the right lung was adherent at its apex, which contained a few old, small, tubercular nodules. The lungs were highly congested with bloody serum. The surfaces of the pericardium were everywhere firmly adherent by old and organized attachments. The roots of the aorta and pulmonary artery were matted together; and so likewise were the auricles. The heart was slightly dilated and hypertrophied; its aortic valves were thickened and slightly shrunken, and thereby rendered incompetent; water, when poured into the aorta, escaped freely through them into the left ventricle. The other valves were quite healthy. The arch of the aorta was dilated, and its internal surface much diseased, exhibiting everywhere signs of fibroid and atheromatous degenerations. In the aortic sinuses were found the openings of three small aneurisms. One, situated above the left coronary valve, admitted the little finger, and led into a pouch which passed behind the pulmonary artery towards the appendix of the left auricle. The openings of the two other aneurisms were at the right of the aorta, and situated above the right coronary valve; one was of the size of a filbert; the other was much larger, and pressed considerably upon the descending cava and the right auricle; the descending cava would not admit the entrance of the little finger.

The arteria innominata was found completely blocked up, for the last inch of its course, by a firm, whitish-red, hardish coagulum, strongly adherent to the walls of the artery, and extending up the whole length of the common right carotid. The opening of the left carotid artery at the aortic arch was not more than one-fourth of its natural calibre; being contracted by hard fibroid exudation into its coats. The left subclavian artery was healthy.

When the dura mater was opened, blood flowed rather freely from the venous sinuses; many red points appeared on the cut surfaces of the brain. A considerable quantity of clear serous fluid was present in the lateral sinuses of the brain. The left carotid artery, where it emerges from

the sella Turcica and passes to the brain, was found completely blocked up by a firm reddish coagulum, which extended for an inch along the middle cerebral artery. The portion of the brain to which the middle cerebral artery—the chief branch of the internal carotid—is directed was much softened, readily breaking down into a pulp, and particularly so was the left corpus striatum. The other parts of the brain, and likewise the vessels at its base, were found quite healthy in appearance.

There was nothing worthy of note to be mentioned concerning the other organs of the body.

Remarks.—This case presents some points of interest both in a physiological and in a pathological sense. It appears clear that the arteria innominata was blocked up at the time of the admission of the patient into the Hospital and for the reasons that the pulse was then imperceptible at the wrist, and that the clot itself was evidently not of recent formation. Consequently, the functions of the brain were duly carried on, notwithstanding that its supply of blood through this artery was cut off, and that not more than one-fourth of the due amount of blood could have passed through the opening of the left common carotid. Also it is evident, that for the last sixty hours of life the only supply of blood which the brain received was that which passed through the left vertebral artery. The cause of the formation of the clots in the arteria innominata, and in the left cerebral artery, must be, to a certain extent, matter of conjecture. There were no local alterations perceptible in the vessels themselves which could give any account of clots. Both vessels, to all appearance, seemed perfectly healthy; the arterial branches at the base of the skull and around the obstructed artery were quite natural to the eye. Perhaps the existence of the aneurisms may give us a clue to the obstruction of the arteries. It is not unfair to presume that it was very possible for some broken-down portions of fibrin to have been washed out of one or other of the aneurisms at the root of the aorta, and to have been carried along

in the current of blood until arrested in their progress, acting then as a ligature of the artery, and causing behind it the formation of a firm fibrinous coagulum. This supposition is strengthened by the fact that the mouths of the aneurisms were large. There were no fibrinous beads around any of the heart's valves, so that the carrying away and arrest of one of these in the circulation could not have caused the clots, after the manner described by Dr. Kirkes. The softening of the brain was clearly secondary to the arrest of the circulation. In this case the supply of blood to the left middle lobe of the brain must have been wholly cut off; there could have been no compensatory supply to it through the right carotid artery.

To the practitioner, also, this case of paralysis, like so many others of its kind, offers a word of fruitful warning. What would a vigorous treatment have done here? What could any interfering treatment do?

CLOTS IN THE PULMONARY ARTERY AND THE HEART.

The late Dr. Scott had suffered from a slight cold for several days previous to the illness which ended in his death. While so suffering, he took an unusually long and fatiguing walk; and on the following day found his right leg stiff, swollen, and painful, the swelling of it being general. Under the influence of rest and fomentations, &c., the swelling gradually abated; and on the fourth day from its commencement I was sent for to see him. It appeared that he had risen from bed, and was engaged in bathing his leg with chloruretted water, when he was suddenly seized with a violent attack of angina; he felt an agonising pain shooting through his sternum, passing to his left shoulder and down his left arm; it seemed "as though his heart must burst." The attack had passed away when I arrived, but his pulse was still very irregular. When he became more tranquil I carefully examined his heart, and besides irregularity and weakness of heart I noticed nothing unnatural

beyond a very slight aortic systolic bruit. He himself had quite recovered his spirits, and was now as cheerful as usual.

I found on inquiry that during the last two years he had suffered from occasional fainting fits and temporary loss of memory, and from other indications of defective power of the heart. He insisted, however, that this present attack of angina arose from his having inhaled the chlorine gas which was given off from the chloruretted bath he used to his leg. He would not admit that it resulted from the effects on his weakened heart of the extra exertion he underwent while bathing his limb. This attack occurred on Monday. On the following Wednesday he was again seized with a similar one; and from this time until his death he was attended by Dr. Walshe and myself.

There now appeared a loud systolic bruit over, and limited to, the region of the right ventricle; and also marked dulness on percussion across the lower part of the sternum. These signs and the general symptoms, and the previous history, left no doubt in the minds of Dr. Walshe and myself as to the existence of a weak (probably fatty) and distended condition of the heart. The bruit also indicated the formation of clots in the heart; and the attacks of angina, it was thought, were possibly associated with passage of clots into the pulmonary artery. Besides, there was distinct evidence of consolidation of the lower lobe of the left lung.

Dr. Scott suffered several similar attacks, and died during the last one, after a most painful and agonising struggle, on the ninth day from the date of the first attack, retaining his senses to the last.

The post-mortem examination fully justified the diagnosis. The heart was much distended with clots; its right ventricle reaching beyond the right border of the sternum; the whole organ was covered with a layer of subpericardial fat, which encroached much on the muscular tissue; the muscular tissue was soft and flabby; the valves were competent,

and no signs of inflammation existed. Portions of the muscular tissue of the heart examined under microscope showed signs of marked fatty degeneration; in a specimen taken from the very centre of the septum ventriculorum, the muscular striæ were scarcely to be seen; and besides this, fatty tissue was found in large abundance around and between the fibres. Little specks of fat were also found beneath the endocardium.

The left pulmonary artery and its two branches were blocked up with a clot, decidedly not of recent formation, which posteriorly was so firmly adherent to the inner coat of artery, as when removed to bring away with it portions of the inner coat. Everywhere throughout the left lung plugs of fibrin projected from branches of the pulmonary artery on the cut surface of the lung. The lower lobe of the lung was solid and black with blood (pulmonary apoplexy); a very few shreds of fibrin on its base, and on the corresponding surface of diaphragm, also indicated pleuritic dry inflammation at those spots: but besides these there were no other signs of inflammation.

The right leg was not examined, but it is not unreasonable to conjecture that there might have been inflammation of the veins of it; and that clots of blood formed in some of them had escaped into the heart, and there laid the foundation of the subsequent fatal mischief. The agonising pain felt along the left costal margin was ascribed by Dr. Walshe to the phrenic pleurisy.

Dr. Scott was fully impressed with the idea, that the attack was associated in some way with the inhalation of the chlorine; and it is worthy of note, that Dr. Richardson, who has experimented with chlorine inhalations, tells me, that he believes the inhalation of the gas is capable, under certain conditions, of producing clots in the heart and pulmonary vessels.

APPENDIX III.

SOUNDS OF THE HEART.

Dr. Halford has, of late years, investigated by experiments the causes of the heart's sounds; and he has arrived at the conclusion: that both of them depend wholly upon the vibrations produced by the tension of the two sets of valves. He supports his views chiefly by the experiment which he has often performed, viz. of exposing the heart of a living animal, and of arresting the flow of blood through it. So long as blood flows through it two sounds are heard, by help of a stethoscope placed on it, but they instantly cease when the flow of blood is arrested. I willingly bear witness to the correctness of this statement; and willingly admit also, that the *chief* cause of the heart's first sound is tension of the auriculo-ventricular valves. But I do not see in what way this experiment invalidates the proposition, that there are other causes which aid in its production. The question resolves itself into this—not whether Dr. Halford stops the sounds of the heart, when he arrests the circulation of the blood through it, but whether he arrests *that first sound which we hear when we listen to the beating heart of a living unmutilated animal?* On the face of the experiment it is evident, that certain of the supposed causes of sound No. 1 can no longer act. There can be no *impulse* against the thoracic walls when they are removed. There can be no *rush of blood through arterial orifices* when no blood is allowed to course through the heart. There can be no true (that is, natural) *bruit musculaire*, where the heart's contractions are, as it were, abortive; for, when no blood enters the heart, there can be no dilatation and no natural systole.

But, on the other hand, when the blood is admitted, *i. e.* when the sounds reappear, then we have these two latter facts in action, viz. rush of blood through arterial orifices, and perfect muscular contractions.

Dr. Halford argues off the other possible causes of heart's first sound in a very unsatisfactory way. As regards the impulse against the thoracic walls (which, by the way, and, alas for the frailty of human ingenuity! was once demonstrated by Majendie to the satisfaction of an admiring body of young *savans*, as the *sole* cause of this first sound), he says it is a mistake altogether, "the blow is only apparent." Now in answer to this physiological view—for it is founded on the assertion that the apex is not tilted forward during systole—I would oppose this simple *pathological* fact, viz. the beat of the heart of a nervous hysterical female, as conclusively demonstrative of the fact, that a heart can rap "like a hammer" against the inside of the thorax. This beat gives us a taste of what it may do in its milder modes. As to the *bruit musculaire*, Dr. Halford does not believe in the existence of the thing at all; but as the fact may at all times be heard over the pectoral muscles of a navvy, and as it is received as true by all Medical Christendom, I cannot admit his exception to the fact. Nor must he say, that in an hypertrophied heart, the first sound should, *therefore* (*i. e.* if it were caused by muscular bruit), be increased; because in such a heart, the other causes, which as here asserted go to the formation of this compound sound, are no longer in perfect action; and because also in hypertrophy the *contractions* are almost invariably more or less imperfect; and so also are the valves themselves. That other argument (which he thinks proves his assertion to demonstration), viz. "that, although muscular action is going on vigorously, no sound is heard," avails nothing for the reason above stated —viz. that inasmuch as there is no blood admitted to the heart's cavaties, there can be neither proper expansion nor proper contraction of the ventricles. If the heart's cavities do not dilate, the muscular fibres cannot return to their natural,

relaxed condition, which precedes contraction. Then, again, to say that the rush of blood out of the ventricles through the arterial orifices produces *no* sound is a palpable error; for it undoubtedly produces the first *arterial* sound—the sound which corresponds with the dilatation of the arteries, and which may be plainly heard over every large artery of the body, and in certain pathological states is heard, as a bruit, even over the radial artery, on the slightest pressure of the stethoscope. The first sound, therefore, as heard over the base of the heart at least, undoubtedly depends in part upon the sudden stretching of the aorta and pulmonary artery. Whether the forcible and sudden expulsion of blood from a cavity like the ventricle through an orifice, which is considerably less in diameter than the aforesaid cavity, produces a sound, is not clear; but it assuredly, *à priori*, appears very probable. The argument used by Dr. Halford—that "he will not admit that the flow of blood either into or out of the heart (in health) can produce sound; for if such were the fact, sound should be developed during the filling of the auricles," &c.—is manifestly a defective one; and for the plain reason, that the flow of blood into the auricles gently, quietly, and equably, is a vastly different affair from the forcible and sudden ejection of blood out of the ventricles into the arteries.

Pathological states of the valves also sustain this position. In some diseased states, for instance, of the mitral valve, we can distinctly hear over the left side of the heart a dull, indistinct sound, which we may fairly suppose to be the first sound *minus* its valvular part. I have heard this sound over the left side of the heart, when the mitral valves have been converted into a rigid cone. And the very difference in the character of the healthy first and second sounds, also, strengthens the argument. They differ not only in duration, but also in tone. If the second be purely membranous, and differs considerably in tone from the first, then we may fairly conclude that the exciting cause is not precisely similar in both.

And it is also worthy of note, that the impulse of the heart and the occurrence of the first sound are not always exactly coincident in time.

We find, moreover, that when the mitral or tricuspid valves are defective, a distinct first sound is rarely heard over the ventricle, whose valves are defective, the sound being, in such case, usually replaced by a murmur. It may, however, be still audible over the aorta and pulmonary artery, and even over the ventricle whose valves are healthy.

When again the sounds are distinct and clear over the left ventricle and the pulmonary artery, but are replaced by a double bruit over the aorta, we are certainly justified in the conclusion, that, at all events, the sounds heard did not arise in the aorta; and when they are replaced by a double bruit over the aorta and left ventricle, but are clearly audible over the pulmonary artery, then the conclusion seems inevitable, that the sounds heard over the pulmonary artery arose in that artery itself.

Further, it may be remarked, that the closure of the auriculo-ventricular valves must, of necessity, be rapid and sudden; but the first sound does not, throughout its whole duration, bear the character of a sound derived from a tense vibrating membrane. It is prolonged, and, in part, heavy, dull, and muffled. And then, again, the presence of a bruit coincident with the heart's systole, does not, invariably, supersede the first sound; for it sometimes happens, that the bruit and the sound are both heard together. Neither are the different acts referred to perfectly simultaneous; the closure of the valves must necessarily precede both the expulsion of the blood from, and the complete contraction of, the ventricles. The expulsion of the blood, too, is not an instantaneous act; the rush of it through the arterial orifices of the heart, though rapidly accomplished, must be, in a restricted sense, a continuous act. The vibration, again, of the coats of the aorta and pulmonary artery, resulting from their distension by the blood forced into them from the

ventricles, is manifestly excited subsequently to the act last mentioned. Hence, then, it appears, that though we commonly speak of the different phenomena which are concerned in, and connected with, the systole of the heart, as occurring simultaneously, yet they in reality follow, by however short an interval, the one upon the other.

Such are the reasons which induce me to adopt the conclusion: that the first sound is chiefly caused by tension of the auriculo-ventricular valves, but that it depends in part upon other causes also.

APPENDIX IV.

IMPULSE OF THE HEART.

Gutbrod's theory, though imperfect as a complete explanation of the heart's impulse, is, I venture to think, well worthy of attention. I therefore give a summary of it.

The following is an explanation of the principles upon which this theory is founded.* When fluids—aëriform or liquid, it matters not which—escape out of any cavity which contains them, either as the natural consequence of their gravity, or through pressure exerted upon the walls of the cavity from without, or from their own expansion within its walls—the equality of the pressure of the fluid upon the inner surface of the cavity is disturbed. The pressure ceases to act—is lost, at that point of the cavity whence the fluid escapes, but is still equally exerted over every other part, and therefore at that part which is opposite to the point whence the fluid escapes; consequently, a less degree of pressure is exerted on the side from which the fluid escapes, than on the opposite side. Now from this inequality of pressure, it necessarily results, that the body containing the fluid tends to move in a direction opposite to that of the escaping fluid.

This tendency is the force which produces the recoil of fire-arms, and which sets in motion what is called Barker's mill; and is the force which, according to the theory we are discussing, is supposed to produce the heart's impulse.

* It is due to Dr. Alderson to state, that, as long ago as 1825, he propounded this explanation of the heart's impulse, and gave illustrations of it, identical to those now offered by Gutbrod and Skoda.

Now careful calculations have been made to explain the force with which the blood escapes from the aortic orifice; and it appears to result therefrom, that the pressure upon every square inch of the inner surface of the left ventricle amounts to about four pounds; and upon every square inch of the right ventricle, to one pound and a half. Hence at each systole, that point of the heart—that is, its apex—which is opposite to the orifices of the aorta and pulmonary artery, will be driven in a direction contrary to that of the currents of blood passing along those vessels, with a force equal to five and a half pounds. The different directions of the two currents naturally tend to move the ventricles into two different axes; but as the muscular power of the left is much greater than that of the right ventricle, the combined movement, thus imparted to the heart, is chiefly in the direction of the stream which issues from the aortic orifice.*

But then comes the question: Is such a force sufficient to account for the impulse? Dr. Davies has answered this, by saying, that there is certainly nothing unreasonable in our believing, that such a force, so rapidly exerted as it is, may impress a movement of recoil on an organ like the heart, loosely suspended, and having an average weight of about ten ounces. This theory, moreover, is not only supported by physical facts, but, as it is asserted, is consistent, in many particulars, with the results of clinical observation.

* All this has been very well worked out by Dr. Davies; "Lectures," etc., p. 230.

APPENDIX V.

RUPTURE OF THE HEART.

The following is an analysis of the cases of rupture of the heart—so called spontaneous rupture—which are recorded in the first seven volumes of the *Pathological Society's Transactions*. The details of these cases strongly confirm the general statement made of the history of rupture of the heart by Dr. Quain, in his well-known paper on Fatty Degeneration of the Heart, recorded in the *Transactions* of the Medico-Chirurgical Society. A few of the cases, indeed, are of the number of those referred to by him in that paper.

Cases of rupture of the heart resulting from external lence — traumatic rupture — related in the Pathological Society's volumes, are of course excluded from consideration here.

Twelve cases of spontaneous rupture of the heart are recorded. In eight of these, the subjects of the disease are said to have suffered from symptoms either of disordered circulation, or respiration, or of both, previously to the occurrence of the rupture. Short breath, pain about the fore part of the thorax, cough, irregular pulse, faintness and vertigo, were the most prominent of these symptoms. Two only are reported to have enjoyed good health. Two had suffered previous attacks of hemiplegia, and in these two intracranial diseases were found after death.

Seven of the cases occurred in the male, and five in the female sex. The average age of the sufferers was about 68 years; the youngest patient was 52, the oldest 79 years old.

In nine cases the rupture opened into the left ventricle of the heart. In four, it was situated in the anterior wall of the left ventricle; in five, in its posterior wall. Thus as far as these few cases are concerned, the opinion, which assigns the anterior wall as being the most ordinary seat of the rupture, is contradicted.

In all the cases the rupture ran in a direction downwards towards the heart's apex. The edges of the rent were generally jagged, and the passage of it through the ventricular walls, oblique. In two cases the rupture opened into both ventricles; in one of them being through the septum ventriculorum, in the other commencing at the septum, and running transversely across the right ventricle for an inch and a half, midway between the auricle and the apex. In all, the part ruptured was that where degeneration of the muscular fibres was most marked. In one instance all other parts of the muscular tissue, excepting that part immediately around the rupture, appeared healthy. In nearly all, if not in all, the coronary arteries were diseased: and in an especial manner was the circulation obstructed through that particular branch, which led to the seat of the rupture.

In two of the cases, temporary hemiplegia had been observed some time before death, and in these cerebral disease was found after death. In one of them was noted the existence of extensive disease of the cerebral arteries at the base of the brain.

Only in one case is the existence of arcus senilis "well developed" recorded. In one case, it is said not to be present. In other cases no reference is made to this point.

Scarcely any information is given as to the condition of the other organs of the body in these cases; and none whatever of the microscopic condition of the smaller cerebral arteries, in those cases, where disease of the brain was found coincident. This is much to be regretted, because, as is now well known, fatty degeneration arising insidiously, and obscurely attacking an organ, is no isolated disorder, but is

an indication of a general morbid influence, which makes itself manifest not in one part alone, but in many parts and organs of the body simultaneously, in a more or less well-marked degree.

The probability is, that disease of the finer cerebral arteries would have been found present in most of these cases had it been searched for; the circulation through them being either obstructed or impeded, just as it is in the case of fatty heart. Indeed, in two of these cases, as we have seen, there had been apoplectic seizures. In a case of fatty degeneration of the heart related by Dr. Quain, where death arose from meningeal apoplexy, the brain was said to be perfectly healthy, and death was attributed to the apoplexy resulting from obstructed flow of blood through the brain, the obstruction being seated in the right ventricle of the heart; but, even in this case, we may be tolerably sure that the finer cerebral vessels were diseased. Mr. Paget first especially called attention to this diseased condition of the smaller vessels of the brain; and his discovery has thrown great light into what had been hitherto a very obscure corner of pathology. What is very significant to our present purpose is this, that the presence of "small granular particles, like molecules of oil," may be frequently observed in the vessels of portions of the brain, which to all appearance are perfectly healthy. A consideration, moreover, of the history of the cases of apoplexy, and fatty degeneration of the heart, independent of its rupture, recorded in these *Transactions*, quite justifies us, at least, in greatly doubting whether mere obstruction to the cerebral circulation situated in the heart, or any violence in the contractions of its left ventricle, is sufficient to produce sanguineous effusion into or upon the brain, unless the cerebral vessels be more or less impaired by disease—be softened or rendered brittle through the presence within their coats of oily or fatty, or earthy matters; and whether, in fact, apoplexy ever occurs unless disease of the cerebral vessels pre-exists.

These points are well worthy of attention, because they

have a practical bearing in relation to the diagnosis of fatty degeneration of the heart. Many of the disordered symptoms (as far as they relate to the cerebral functions) hitherto attributed to the defective state of the heart, and therefore assigned as indicators of its diseased state, may in reality be signs of disease seated in the brain, of softening of its substance, or degeneration of its vessels. The fatty state of the heart, and the diseased condition of the brain, in all probability, run coincidentally, and we should, therefore, be very cautious in drawing conclusions from the above set of symptoms, as to their seat of origin being fixed exclusively in either the one or the other organ—in the heart, or in the brain.

The facts contained in these volumes teach us to give a very wide signification to these fatty degenerations of tissues; and thus, for instance, to see a very close connexion between the pathological conditions which occasion sanguineous apoplexy and rupture of the heart. In both cases, we find local disease of the vascular system, and in both alterations in the textures of the parts supplied by the altered vessels; the rupture in either case resulting from a softening and disintegration of the parts thus locally affected. The difference in the symptoms produced has a distinct relation to the nature and function of the organ, impeded in its actions by the sanguineous effusion, the result of the accident. Much stress should be laid upon these particulars, because it now becomes clear, that we cannot (during life at least) study the symptoms of fatty degeneration of the heart, without taking into account the possible, or rather probable coexistence of alterations of the cerebral structures, as well as of diseased states of other organs and parts of the body.

The history of the seven cases of apoplexy—contained in five of the volumes of the *Transactions*—of sanguineous effusion into or upon the cerebral structures, fully confirms these views. In all of these, disease of the vessels at the base of the brain was observed, except in one case, where their

condition is not alluded to. The arteries were "studded with atheromatous deposits," were "ossified," were "extremely degenerated." In two instances the vessels around the injured portion of the brain were microscopically examined, and signs of degeneratien were observed in them, their coats containing fatty or calcareous particles. Four of the cases of apoplexy occurred in males, three in females; their average ages being about 50—the youngest was 34, the oldest 91 years of age. In all of them disease of some one or other part of the body was observed—granular kidney, fatty heart, enlarged heart, atheroma of the aorta; but here, as in the history of the ruptures of the heart, the condition of the different organs of the body is in but few of the cases clearly and specifically given. In two of the cases the effusion was superficial; in eleven the blood escaped into the cerebral substance.

THE END.

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